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INSTRUMENTS AND TECHNICS FOR THE CLINICAL TESTING OF LIGHT SENSE

I. REVIEW OF THE RECENT LITERATURE

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One of the most fundamental problems of physiologic optics is the study of the light sense, or achromatic sensitivity of the eye, and in particular the increase in sensitivity which occurs when the eye is exposed to darkness. The term photopic vision is used to designate the condition of the eye when exposed to bright light; scotopic vision, to designate the condition of the eye when exposed to darkness or to a very dim illumination. It is generally believed that photopic vision is dependent chiefly on the retinal cones and scotopic vision on the rods.

In the usual ophthalmologic examination the subjective tests include determination of the central visual acuity and examination of the visual fields with white and colored test objects. Study of the visual fields with white test objects is essentially a somewhat crude method of testing the light sense throughout the retina. Since, however, this and the other subjective tests are applied as a rule only to the light-adapted eye, they are tests primarily of photopic vision. For a complete study of the functional capacity of the eye, similar tests should be applied to the dark-adapted eye.

The tests of scotopic vision most frequently used for clinical studies are either (*a*) determination of the light minimum of the eye after exposure to darkness for a specified time or (*b*) determination of the rate of dark adaptation. The latter involves measurements of the light minimum at successive intervals during exposure to darkness until there is little or no further increase in sensitivity with continued stay in the dark. A review of the various clinical procedures which have been proposed for the study of scotopic vision shows that in many only a single region of the retina is tested and that none will give a general survey of the entire retina, such as is provided for photopic

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vision by detailed perimetric studies. Moreover, because of the especial difficulty of controlling the fixation of the dark-adapted eye, it is often impossible to determine for a given instrument and technic just what region of the retina is actually tested.

The first quantitative studies of dark adaptation were made by Aubert in 1865.[1] Many of the early investigations following that of Aubert were devoted to a study of the variable factors affecting the sensitivity to light in the normal eye. Other studies investigated the effect on the light sense of various pathologic conditions of the eye. With these two lines of investigation as a foundation, later reports were devoted to the problems of developing a standardized clinical test for some aspect of the light sense and of determining the possible diagnostic and prognostic value of the test in different pathologic conditions of the eye.

The literature published prior to 1929 dealing with light sense and with dark adaptation in particular has been reviewed in detail by Adams.[2] In the present review of the literature papers published prior to 1929 have therefore been excluded for the most part. This review is likewise limited chiefly to those reports devoted to the clinical rather than to the theoretic aspects of the subject.

In evaluating the merits of the different instruments and procedures proposed for the clinical testing of dark adaptation, the following requirements of an ideal test should be kept in mind. (*a*) A preexposure field of constant brightness is desirable in order that the initial level of adaptation of the eye may be standardized. (*b*) Means should be provided for illuminating the test field with light of a known composition, variable in intensity by known amounts over a wide range. (*c*) Since retinal illumination increases with the area of the pupil, there should be some method of eliminating or correcting for this variable factor. (*d*) Means should be provided for the control of fixation, since the light sense varies in different regions of the retina. (*e*) In order to distinguish between pathologic and normal sensitivity to light, the range of variation in a sufficiently large group of nonpathologic eyes should be known.

Among the most extensive of the recent clinical studies in this field are those published by Derby and his co-workers [3] at the Massachusetts

1. Aubert, H.: Physiologie der Netzhaut, Breslau, E. Morgenstern, 1865.

2. Adams, D.: Dark Adaptation: A Review of the Literature, Medical Research Council, Special Report Series, no. 127, London, His Majesty's Stationery Office, 1929.

3. (*a*) Waite, J. H.; Derby, G. S., and Kirk, E. B.: The Light-Sense in Early Glaucoma, Particularly the Achromatic Scotopic Threshold at the Macula: A Preliminary Report, Tr. Ophth. Soc. U. Kingdom **45**:301-331, 1925. (*b*)

Eye and Ear Infirmary. During the period between 1924 and 1933, members of this group published a series of articles dealing with (*a*) apparatus and technic for measuring the curve of dark adaptation, (*b*) the range of variation of the adaptation curve in normal eyes and (*c*) the adaptation curve in the glaucomatous eye.

The two forms of apparatus described by Waite, Derby and Kirk [3a] in 1925 and by Derby, Chandler and Sloan [3d] in 1929 fulfil the first three requirements outlined. The intensities of light are specified in absolute units. The variable effect of the size of the pupil is eliminated as follows: The pupil is fixed with pilocarpine, its diameter measured and a correction factor applied to the experimentally determined values of light minimum in order to convert these into units of retinal illumination.[4] In the method of Derby and his co-workers the correction factor for size of the pupil is applied only to the experimentally determined values of the light minimum. Although the preexposure field is maintained at a constant physical brightness, the resulting retinal illuminations thus vary with the size of the subject's pupil. The consequent differences in the level of light adaptation of different patients probably have some effect on the determinations in the early stages of dark adaptation but have no doubt only a negligible effect on the values of the light minimum after ten minutes or more of dark adaptation.

Derby, G. S.; Waite, J. H., and Kirk, E. B.: Further Studies on the Light Sense in Early Glaucoma, Arch. Ophth. **55**:575-579, 1926. (*c*) Derby, G. S.; Chandler, P. A., and O'Brien, M. E.: Further Studies on the Light Sense in Early Glaucoma, Tr. Sect. Ophth., A. M. A., 1928, pp. 37-51. (*d*) Derby, G. S.; Chandler, P. A., and Sloan, L. L.: A Portable Adaptometer, Tr. Am. Ophth. Soc. **27**:110-130, 1929. (*e*) Casten, V., and Shaad, D. J.: Diagnostic Value of Tests of the Light Sense in Early Glaucoma, Arch. Ophth. **9**:52-55 (Jan.) 1933.

4. In making this correction it is assumed that the intensity of illumination in the retinal image is directly proportional to the area of the pupil. The validity of this assumption has been questioned by Stiles and Crawford (Luminous Efficiency of Rays Entering the Eye Pupil at Different Points, Proc. Roy. Soc., London, s.B **112**:428-450, 1933) on the basis of experiments showing that rays passing through the periphery of the pupil are less effective than rays passing through the center. In these experiments the principle of the Maxwellian view, in which the incident light converges to a small image in the plane of the observer's pupil, was used. It was found that the apparent brightness of the field was at a maximum when the image was formed in the center of the pupil and decreased gradually with a shift of the image from the center to the periphery. The data do not apply directly to the more usual conditions, in which the test field is diffusely illuminated and its image is focused on the retina by means of the refracting system of the eye. The results reported by Stiles and Crawford suggest, however, the desirability of further experiments to determine whether or not under the latter conditions of observation the apparent brightness of the retinal image may without appreciable error be assumed to be proportional to the area of the pupil.

The test field is a vertical oblong, 4 by 2 degrees in size. The patient is instructed to fixate the center of this field. Waite, Derby and Kirk stated:

> If the patient gazes directly at the position of the light (pointed out in advance) until the light itself becomes visible, he gives a true foveal threshold, which is high. On the other hand, if the patient does not follow instructions during the first ten minutes, he looks in whatever direction he can best see the light, and hence gives a parafoveal or rod threshold which is low. In either case, after the first 15 minutes of dark adaptation have brought the sensitivity of rods and cones nearer to equality, the direction of the gaze does not alter the threshold.

The authors' assumption that foveal and parafoveal sensitivity are more nearly equal in dark than in light adaptation is the reverse of the generally accepted view. The experiments of Breuer and Pertz, for example, reported in 1897 by von Kries,[5] show that in the dark-adapted eye the relative insensitivity of the foveal region is marked. Wölfflin[6] in 1910, using a test field 3 degrees in diameter and a red light as a fixation control, found that during the first half minute of adaptation to darkness the central sensitivity is greater than the paracentral but that after one minute of dark adaptation the central sensitivity is definitely less than the paracentral.

In 1928 Derby, Chandler and O'Brien [3c] published data showing the range of variation in the adaptation curves of a group of normal subjects. The number of eyes tested is not stated. The curve designated as the upper limit of normal shows values of the light minimum from 1.8 to 2.2 times the median values. Members of the same group also reported studies of the adaptation curve in glaucomatous eyes (Waite, Derby and Kirk, 1925; Derby, Waite and Kirk, 1926; Casten and Shaad, 1933). As a result of the earlier investigations, reported in 1925 and 1926, they believed that the test would prove of definite value in the diagnosis of glaucoma. A final evaluation of the entire study, covering the period between 1925 and 1933 and based on successive tests of over 400 patients, was reported by Casten and Shaad in 1933. They concluded that the test of the light sense is not of great diagnostic value except perhaps as a confirmatory test and is also of little prognostic value. Normal adaptation curves were obtained for some patients with definitely established glaucoma. Twelve of the 40 patients in whom glaucoma developed in the second eye during the course of these studies had an abnormal adaptation curve as the first symptom. In the remaining 28 patients, elevated tension, field defects or pathologic changes in the nerve heads were noted before any pathologic changes in the adaptation curve were observed. It was found that patients

5. von Kries, J.: Ueber die absolute Empfindlichkeit der verschiedenen Netzhautteile im dunkeladaptierten Auge (Nach Versuchen der Herren Dr. Breuer und A. Pertz), Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **15**:327-351, 1897.

6. Wölfflin, E.: Ueber Dunkeladaptation von fovealen und parafovealen Netzhautpartien, Arch. f. Ophth. **76**:464-477, 1910.

with established primary glaucoma showed on repeated tests of the light sense a much greater random variability than did normal observers.

Delaney [7] in 1930 proposed a simplified clinical test of the light minimum of the dark-adapted eye. Measurements are made by means of Tscherning's photometric glasses of the light minimum after one-half hour of dark adaptation. A standard wax candle is used as the source of light. From preliminary experiments he concluded that control of the preexposure of the eye is unnecessary, since the variations due to different levels of previous light adaptation are of the same magnitude as the normal daily variations in sensitivity of the eye. Standardization of the size of the pupil was deemed unnecessary for the same reason.

Tests of a group of normal eyes showed an average value of light sensitivity of 8.5 to 8.75 (expressed in Tscherning's logarithmic unit, the photopter [8]). The borderline limit of normal is taken as 8 photopters. This value is equivalent in arithmetic units to a light minimum of 3 to 6 times the average value. The number of normal eyes tested was not stated. Delaney expressed the belief that some of the variability in the results is to be attributed to difficulties in fixation caused by the relative central scotoma of the normal dark-adapted eye. The region of the retina tested was assumed by Delaney to be parafoveal except in some patients of low intelligence who tend to take a foveal rather than an excentric fixation.

A marked reduction in sensitivity to light was found in 4 patients with retinitis pigmentosa and in 1 patient with retinitis punctata albescens. The patients with retrobulbar neuritis, optic neuritis, atrophy of the optic nerve and glaucoma showed only a slightly reduced sensitivity to light, although advanced defects in the visual fields were present in most cases. The 9 patients with questionable early glaucoma had normal values of the light minimum.

Delaney expressed the belief that although the test contributes little aid in diagnosis, it is nevertheless of value, particularly in conditions such as retinitis pigmentosa, in measuring defects in sensitivity to light which might prove an economic handicap in certain occupations.

In 1931 Ferree and Rand [9] described an apparatus, somewhat similar to that of Derby and his co-workers, for the determination of the light minimum and the adaptation curve. In this instrument both the preexposure field and the test field are viewed through an artificial pupil 1.5 mm. in diameter. Consequently the apparent brightness of both the preexposure and the test field are uninfluenced by differences

7. Delaney, J. H.: Light Sense as Tested by Photometric Glasses of Tscherning, Am. J. Ophth. **13**:1058-1063, 1930.

8. The glass numbered 1 on the photopter scale transmits $\frac{1}{10}$ of the incident light; that numbered 2 transmits $\frac{1}{100}$ of the incident light, etc.

9. Ferree, C. E., and Rand, G.: A New Type of Instrument for Testing the Light and Color Sense, Am. J. Ophth. **14**:325-333, 1931.

in the size of the pupil in the different patients. The test field is a horizontal oblong, 10 by 3 degrees in size. As with Derby's technic, an attempt is made to test the macular region by instructing the patient to fixate the center of this field. Ferree, Rand and Stoll [10] (1934) made the following statement with regard to control of fixation:

> Little or no difficulty was experienced in the control of fixation for a stimulus of this size as its edges extended into the more sensitive peripheral retina and there was in consequence no incentive to take an excentric fixation, *i. e.*, the stimulus was most clearly seen when its center was fixated.

Using the aforementioned apparatus and procedure, these investigators (1934) obtained norms for 206 nonpathologic eyes. They found a fairly consistent increase in light minimum with age and a differentiation between the results for subjects above and below 35 years of age. Separate norms are therefore given for these two age groups. The borderline normal values of the light minimum are from 2 to 4 times the median values. No determinations of the light sense in pathologic conditions of the eye made with the Ferree and Rand instrument have been published.

Matthey [11] in 1933, using the Nagel adaptometer, determined adaptation curves for 54 normal eyes. The preexposure was standardized by a ten minute period of light adaptation. In order to eliminate the variable effect of the size of the pupil, the pupils were dilated with a solution of homatropine and cocaine. The diameter of the circular test field subtended 10 degrees. A red light located 10 degrees below the center of the test field was used as a fixation object. The borderline normal curve proposed by Matthey is that which differs from the average normal by 3 times the standard deviation. The borderline values of the light minimum obtained in this way are from 2.6 to 5.6 times the average normal for the same interval of dark adaptation.

In the same year Accardi [12] reported the results of a series of clinical investigations with the Nagel adaptometer. Determinations of the thresholds were made after thirty minutes of dark adaptation. The patient was kept in a lighted room for about one-half hour before entering the dark room. Differences in the intensity of illumination to which the patient was preadapted were found to have a negligible effect on the thresholds after thirty minutes in the dark. A red light

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10. Ferree, C. E.; Rand, G., and Stoll, M. R.: Critical Values for the Light Minimum and for the Amount and Rapidity of Dark Adaptation, Brit. J. Ophth. **18**:673-681, 1934.

11. Matthey, G.: Eine "Standardkurve" der Dunkeladaptation für klinische Untersuchungen, Arch. f. Ophth. **129**:275-298, 1932.

12. Accardi, V.: La soglia dell'eccitamento luminoso in condizioni normali e patologiche e sotto l'azione di alcuni farmaci, Ann. di ottal. e clin. ocul. **61**:561-611, 1933.

placed above the test field was used to control the direction of fixation. The results were not corrected for differences in the size of the pupil.

From his measurements on normal eyes, Accardi found an average normal sensitivity of 35 and a normal range of from 65 to 14.9. (Sensitivity is proportional to the reciprocal of the light minimum.) The borderline limit between normal and pathologic values of the light minimum is thus about 2.3 times the average value of the light minimum.

From determinations of the light minimum in a number of different pathologic conditions, Accardi concluded that the test is of diagnostic value particularly in metasyphilitic conditions and in the early stages of glaucoma. The patients with tabes, the tabetic form of dementia paralytica and similar conditions showed reductions in sensitivity to light which he believed were of a greater degree than could be explained as due to the presence of small pupils. In the case of the patients with glaucoma, since there was an increase rather than a decrease in sensitivity when a miotic was used, the subnormal values of the light sense could not be attributed to the presence of contracted pupils.

In 1934 Spaeth [13] described an apparatus for the clinical determination of the light minimum and reported the results of tests on a group of patients with various pathologic conditions. The technic of the test employed is as follows: Especially constructed light-proof goggles are worn by the patient. The light stimulus is obtained by holding a standardized source of light directly in front of perforations in the goggle. The illuminated test field thus obtained subtends an angle of approximately 72 degrees. Variations in the brightness of the test field are produced by means of a pair of glass prisms mounted in a carrier attached to the goggle. As the prisms are separated, the thickness of the glass is decreased, with a consequent increase in the amount of light transmitted. The results of the determinations of the thresholds are expressed in terms of an arbitrary scale described by the author in these words:

> The greater the number the more nearly perfect is the patient's light minimum; the lower the number the greater is his light threshold, and hence his light sense defect; for example, from 1.5 to 2.5 is a very satisfactory light threshold, while from 0.01 to 0.001 would be a very high and pathologic light sense threshold.[14]

Spaeth recognized the need for correcting the measurements for differences in pupillary area. Before the determinations of the thresholds were made, the subject's pupil was fixed to light by the

13. Spaeth, E. B.: Clinical Determination of the Light Threshold, Arch. Ophth. **11**:462-485 (March) 1934.

14. A spectral transmission curve of the glass used in the prisms is given. It shows marked selectivity, with a maximum transmission in the green. In view of the selective transmission of the glass, considerable change in composition of the stimulus light must occur with change in the thickness of the glass. For this reason, any specification of the light stimulus in quantitative terms would be extremely difficult.

use of a mild mydriatic or miotic; the diameter was then measured. An empirically determined correction factor based on the size of the pupil was applied to the measurements of the thresholds. The fact that the intensities of light were not expressed in quantitative units, however, makes any accurate correction for size of the pupil impossible. The results of the determinations of the light sense for a group of patients with various pathologic conditions of the eye are reported. The findings, Spaeth said, though not sufficiently extensive for final conclusions, suggest that the test will prove of clinical value.

A group of Italian investigators, Lo Cascio, Hertel, Cucchia and Zanettin, have reported studies of the light sense which differ from most other recent studies in that they emphasize the comparative sensitivities to light of different regions of the retina.

Hertel [15] in 1930 described an adaptometer made in the form of an illuminated hollow sphere. In devising his instrument and technic, he recognized the need for specifying the intensities of light in absolute units and for controlling the previous state of adaptation of the eye, the region of the retina tested and the area of the pupil. With his instrument he determined the adaptation curves of 10 normal subjects for the fovea and for an extrafoveal region of the retina. Hertel pointed out that there was need for further study of the range of individual differences in sensitivity to light.

Cucchia [16] in 1928 and Zanettin [17] in 1931 reported clinical studies in which they used the photometer devised by Lo Cascio [18] in 1924. With this instrument the light sensitivity can be measured at the fovea, at 30 and 50 degrees from the fovea in the horizontal meridian and at 20 and 40 degrees from the fovea in the vertical meridian. The test field subtends an angle of 2 degrees. The region of the retina tested is controlled by the use of a luminous fixation point.

Cucchia studied with this instrument the light sensitivity of patients with various optic neuropathies. Measurements were made in the dark-adapted eye of the sensitivity at the fovea and at eight points in the peripheral field. The size of the pupil was not controlled. The 28 persons with pathologic conditions included patients with neuroretinitis,

15. Hertel, E.: Il senso luminoso: II. Esame del senso luminoso con un addattometro a sfera quale mezzo per ottenere l'unificazione, Ann. di ottal. e clin. ocul. **58**:643-675, 1930.

16. Cucchia, A.: Ricerche cliniche sul comportamento del senso luminoso centrale e periferico nelle affezioni del nervo ottico, Ann. di ottal. e clin. ocul. **56**: 385-402, 1928.

17. Zanettin, G.: Comportamento del senso luminoso centrale e periferico nel glaucoma cronico semplice, Ann. di ottal. e clin. ocul. **59**:847-882, 1931.

18. Lo Cascio, G.: Appareccio per l'esame del senso luminoso nelle parti periferiche della retina nell'occhio umano, Soc. ital. di oftal. **10**:27-30, 1925.

chorioretinitis, atrophy of the optic nerve, papilledema and retrobulbar neuritis. The results were compared with those for 2 subjects with normal eyes. Cucchia concluded that the degree of reduction in sensitivity to light depends more on the severity than on the nature of the involvement of the optic nerve. He found the relative distribution of sensitivities in different parts of the retina to be the same in the normal and in the pathologic eyes except for 1 patient. This patient had a central scotoma and showed a greater decrease in sensitivity in the central than in the peripheral portion of the retina.

Zanettin made similar studies of 17 patients with chronic simple glaucoma. The results were compared with the average values for 10 normal persons. He pointed out that the glaucomatous patients who were using pilocarpine would have, because of the small pupil, somewhat higher thresholds than normal subjects, but he expressed the belief that the increase in threshold due to this factor would not be great. He concluded that a reduction in the light sense is an early symptom of glaucoma. The earliest and most frequent defect in sensitivity to light was found in the temporal portion of the retina (nasal meridian of field).

In 1934 Feldman [19] described an apparatus for the determination of the adaptation curve which, like those of the Italian investigators, has provisions for testing the light sense in different retinal regions. With this instrument the test field can be either a circular area subtending at the eye an angle of 30 degrees or a rectangular strip 30 degrees in width and 3 degrees in height. A small red fixation light is placed at the edge of the test field. By rotating the test field to different positions it is possible to test an area of the retina to the right, to the left, above or below the fixation point. A preexposure field of fixed brightness is used to standardize the previous level of adaptation. In the preliminary investigations with this instrument an artificial pupil was used. This was found to be unsatisfactory, and the procedure used by Derby and his co-workers to correct for size of the pupil was later substituted. With this apparatus Feldman [20] determined the adaptation curves of 178 patients with normal eyes, 68 of these having no refractive error. He gave numerical data on the normal range only for the values of light minimum after fifteen minutes of dark adaptation. The average normal light minimum was found to be 0.00001 photon; the borderline between normal and pathologic light minimum, 0.00002 photon. Thus the borderline normal value is twice the average normal.

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19. Feldman, J. B.: Instrument for Determining Course of Dark Adaptation and for Measuring Minimum Light Threshold, Arch. Ophth. **12**:81-85 (July) 1934.

20. Feldman, J. B.: Dark Adaptation as a Clinical Test: Technic and Results, Arch. Ophth. **15**:1004-1019 (June) 1936.

Determinations were also made by Feldman of the curves of dark adaptation of a number of patients with glaucoma, retinitis and pigmentary degeneration of the retina and of a few persons with other miscellaneous diseases of the eye. For some patients different parts of the retina were tested by changing the relative positions of the fixation point and the test field. The author stated that "by means of the variable sector and accurate fixation, the entire retina was examined and found to show an equal sensitivity throughout in health and disease."

Of the 109 patients with glaucoma, 8 showed normal adaptation curves and the remaining showed pathologic curves. Four of the 6 patients with retinitis showed pathologic curves. The 4 patients with pigmentary degeneration of the retina showed pathologic adaptation curves. Three patients with atrophy of the optic nerve all showed normal curves. The author concluded that the test is of definite value in the study of ocular disease.

In a third paper, published in 1937, Feldman [21] reported further studies of dark adaptation, chiefly of patients without retinal disease but with conditions such as diseases of the blood or of the blood vessels and diseases related to vitamin A deficiency. A pathologic graph of dark adaptation was obtained for a majority of the patients with arteriosclerosis. The results for patients with blood disease were found to be dependent on the basic cause of that condition. The dark adaptation graph was pathologic for the 4 patients who were suspected of having a vitamin A deficiency.

In 1937, also, Ezickson and Feldman [22] reported studies of the curves of dark adaptation of a group of 25 patients with urinary lithiasis and of a control group of 50 patients free from urinary calculi. All but 1 of the 25 patients with urinary lithiasis had a pathologic curve of dark adaptation. Of the control group, 46 patients showed normal dark adaptation. The pathologic dark adaptation of 4 patients was associated with jaundice (2 cases), myxedema (1 case) and juvenile diabetes (1 case). Fifteen of the 24 patients with pathologic dark adaptation, retested after from six to nine months of vitamin A therapy, did not show any improvement. It was suggested that the pathologic dark adaptation was probably due to lack of assimilation or utilization of vitamin A and that this condition and urinary lithiasis may have a common metabolic basis.

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21. Feldman, J. B.: Dark Adaptation as a Clinical Test: Further Studies, Arch. Ophth. **17**:648-661 (April) 1937.

22. Ezickson, W. J., and Feldman, J. B.: Signs of Vitamin A Deficiency in the Eye Correlated with Urinary Lithiasis: Report of Clinical Studies and Investigations on Twenty-Five Patients, J. A. M. A. **109**:1706-1710 (Nov. 20) 1937.

In the same year Feldman [23] reported a simpler form of test for dark adaptation, which measured the time of dark adaptation required to see a dimly illuminated test field of fixed brightness. If the test field was seen after five minutes or less of dark adaptation, the patient was considered to have a normal adaptation to darkness. The previous level of light adaptation of the eyes was standardized as in the more elaborate instrument. In the simplified form of the test no attempt was made to standardize or correct for differences in size of the pupil. Both eyes were tested together. Approximate control of fixation was obtained by "indicating occasionally with the subject's hand where the light stimulus (the arrow) is to be seen."

Of the 116 patients examined, 47 showed pathologic dark adaptation. The reduced light sense of some of these patients was attributed to some pathologic condition, such as choroiditis, high myopia, arteriosclerosis, renal calculi, sinus infection or thyroid disease. The reduced light sense of others, with irrelevant histories, was presumed to be due to vitamin A deficiency without subjective symptoms. Several patients with pathologic ocular conditions affecting one eye only showed normal dark adaptation. Three patients with bilateral choroiditis also had normal light thresholds. Feldman suggested in explanation of the latter findings that small areas of localized choroiditis may fail to be included in the area the light sense of which is tested.

There is an extensive literature on the subject of vitamin A deficiency, its clinical manifestations, the relationship between vitamin A and scotopic vision and its bearing on visual theory. These papers have been reviewed in detail by Jeghers.[24] I shall consider only those reports which have a bearing on the general problem of the clinical testing of light sense and dark adaptation.

Prior to 1937, Jeans and his co-workers,[25] Park,[26] Snelling,[27] Jeghers,[24] Maitra and Harris [28] and other investigators used the Birch-

23. Feldman, J. B.: An Instrument for Qualitative Study of Dark Adaptation, Arch. Ophth. **18**:821-826 (Nov.) 1937.

24. Jeghers, H.: Night Blindness as a Criterion of Vitamin A Deficiency: Review of the Literature with Preliminary Observations of the Degree and Prevalence of Vitamin A Deficiency Among Adults in Both Health and Disease, Ann. Int. Med. **10**:1304-1334 (March) 1937.

25. Jeans, P. C., and Zentmire, Z.: (*a*) A Clinical Method for Determining Moderate Degrees of Vitamin A Deficiency, J. A. M. A. **102**:892-895 (March 24) 1934; (*b*) Prevalence of Vitamin A Deficiency Among Iowa Children, ibid. **106**: 996-997 (March 21) 1936.

26. Park, I. O.: Preliminary Observations on Vitamin A Deficiency as Shown by Studies with the Visual Photometer, J. Oklahoma M. A. **28**:357-364, 1935; Further Observations on Vitamin A Deficiency as Shown by Studies with the Visual Photometer and Clinically, ibid. **29**:129-141, 1936.

27. Snelling, C. E.: A Study of the Birch-Hirschfeld Photometric Test for Vitamin A Deficiency, J. Pediat. **9**:655-661 (Nov.) 1936.

(Footnotes continued on next page)

Hirschfeld photometer to study vitamin A deficiency on the assumption that in the absence of other pathologic ocular conditions a subnormal light sensitivity, whether manifest after light adaptation of the eye or after ten minutes of dark adaptation, is an indication of vitamin A deficiency.

The test field of the Birch-Hirschfeld photometer consists of five illuminated spots in the form of a quincunx. Two independent means of varying the brightness of the test field are provided: an iris diaphragm and an absorbing wedge. Because of the decrease in transmission of the wedge from left to right, the two right hand test spots are slightly dimmer than the center spot, and this in turn is dimmer than the two left hand spots. The technic used in the various investigations is in general as follows: Before determinations of the thresholds are made, the patient's eyes are adapted to a fixed high level of illumination. The threshold intensity is taken as the lowest intensity at which just three of the five spots are seen. The determinations are made "as soon as possible" after the bright light is turned off and again after ten minutes in the dark. These determinations give two points on the adaptation curve, the first after a short interval of dark adaptation and the second after adaptation is almost complete. Because of the rapid increase in sensitivity at the beginning of dark adaptation, the first reading may show marked variation with only slight differences in the time at which the measurement is made. The readings after ten minutes of dark adaptation are less influenced by slight variations in the actual time at which the determination is made, because the increase in sensitivity is more gradual at this stage of dark adaptation. The thresholds are as a rule recorded in arbitrary values in terms of the settings of the diaphragm and the wedge. No correction is made for differences in size of the pupil. As a rule both eyes are tested together. Each of the investigators determined for his own instrument and technic the range of normal readings by means of tests on a small group of adults known to be on an adequate diet. Jeans and his co-workers, Park, Maitra and Harris, and Jeghers all found that in an unselected group of subjects a large number showed subnormal sensitivity to light either on the basis of the initial readings or on the basis of those made after ten minutes of dark adaptation. In all four of the investigations it was found that when subjects with subnormal light sense were put on vitamin A therapy a majority showed subsequently an improvement or a restoration to normal. Maitra and Harris were, however, the only investigators who tested a control series of patients with subnormal light sense who were also given repeated tests but who received no vitamin

28. Maitra, M. K., and Harris, L. J.: Vitamin A Deficiency Among School Children in London and Cambridge, Lancet 2:1009-1014, 1937.

A therapy. Of 40 patients with subnormal light sense prior to vitamin A therapy, 75 per cent became normal, 22.5 per cent improved and 2.5 per cent showed no change after four weeks of treatment. Of the control group of 20 patients who received no treatment, none became normal, 10 per cent improved and 90 per cent showed no change.

Snelling made similar studies with the Birch-Hirschfeld photometer and believed at first that his results confirmed the findings of Jeans and others with regard to the frequency of mild degrees of vitamin A deficiency, 53 (32 per cent) of his 145 subjects showing poor dark adaptation when compared with the control group of 15 normal adults on a good diet. Twenty-eight of those with subnormal light thresholds were retested after receiving cod liver oil daily for from two to six weeks, and 18 of these showed an apparent improvement. In later experiments the wedge and the iris diaphragm were calibrated so that the results might be expressed directly in intensity units. When the previous data for the control group and the 145 unselected subjects were recomputed, it was found that only a few of the latter actually had higher thresholds than the control group. After further studies, Snelling concluded that the Birch-Hirschfeld photometer was unsatisfactory for the estimation of small variations in dark adaptation because of the lack of reproducibility of the results in successive tests.

A modification of the Birch-Hirschfeld instrument, known as the biophotometer, was described by Jeans, Blanchard and Zentmire [29] in 1937. In the latter instrument the preexposure field forms an integral part of the apparatus. A rheostat in series with the light source is used to regulate the brightness of the test field. The same five point form of test field is used, the gradation in brightness from left to right being obtained by the use of filters of different transmissions. The thresholds are expressed in milli-footcandles. As in the previous investigations made by Jeans and his co-workers with the Birch-Hirschfeld photometer, both eyes are tested together, and no correction is made for differences in the size of the pupil among the different persons tested. Jeans and his co-workers gave tentative values for normal, borderline and subnormal readings on the instrument. Later in the same year Jeghers [30] made a more extensive study of the range of the thresholds of normal adults. From a group of 100 medical students free from serious ocular disease, the 50 showing the highest sensitivity to light (i. e., the lowest thresholds) were taken as the normal group. The initial thresholds taken immediately after three minutes' preexposure to the brightly lighted field varied for the normal

29. Jeans, P. C.; Blanchard, E., and Zentmire, Z.: Dark Adaptation and Vitamin A: A New Photometric Technic, J. A. M. A. **108**:451-458 (Feb. 6) 1937.

30. Jeghers, H.: The Degree and Prevalence of Vitamin A Deficiency in Adults, with a Note on Its Experimental Production in Human Beings, J. A. M. A. **109**:756-762 (Sept. 4) 1937.

group from 0.3 to 0.7 milli-footcandle. The threshold readings after ten minutes of dark adaptation varied between 0.0148 and 0.122 milli-footcandle.

A third clinical study in which the biophotometer was used is that of Corlette, Youmans, Frank and Corlette.[31] These investigators tested two groups of patients. Group 1 was composed of physicians, technicians and clerical workers who were presumably healthy and on an adequate diet. Group 2 was composed of 50 adult clinic patients with either functional or minor organic illnesses, many of whom gave histories of inadequate diets. Patients with marked refractive errors or ophthalmoscopically demonstrable ocular lesions were excluded. Eleven subjects in group 1 and 22 in group 2 showing a relatively poor dark adaptation were retested after receiving vitamin A therapy, and all but 1 showed improvement. A similar procedure was carried out for 10 subjects showing good dark adaptation on the first test. The average values of the light minimum for both groups showed a decrease

TABLE 1.—*Average Threshold Values*

Time of dark adaptation in minutes	0	3	6	10
Thresholds in milli-foot candles	(initial reading)			
33 patients with high thresholds				
Before vitamin A therapy	1.052	0.264	0.1084	0.0577
After vitamin A therapy	0.5365	0.179	0.0826	0.033
11 patients with low thresholds				
Before vitamin A therapy	0.384	0.072	0.030	0.013
After vitamin A therapy	0.310	0.064	0.027	0.009

after vitamin A therapy, the more marked changes occurring in the group of 33 patients with high values of the light minimum. The average threshold values, computed from tables 3, 4 and 5 of the original article, are given here in table 1.

In order to study the reproducibility of the test results 3 subjects with low thresholds, i. e., with relatively good dark adaptation, were retested on ten successive days. No change was made in their diet. The results of the first and second tests taken from table 6 are shown in table 2.

No tests were made of reproducibility in patients showing initially a relatively poor dark adaptation. It is conceivable, however, that the high thresholds of such patients might be due to poor cooperation and that improvement in a second test would be more likely to occur than when the first test showed good dark adaptation.

Jeans, Jeghers and Corlette and his co-workers concluded from their respective studies with the biophotometer that the instrument and

31. Corlette, M. B.; Youmans, J. B.; Frank, H., and Corlette, M. G.: Photometric Studies of Visual Adaptation in Relation to Mild Vitamin A Deficiency in Adults, Am. J. M. Sc. **195**:54-64, 1938.

technic provide a satisfactory method for the detection of subclinical vitamin A deficiency. The conclusions reached in two recent reports, however, do not support this view. Palmer and Blumberg [32] tested 585 school children with the biophotometer and found that on successive tests the percentage showing subnormal readings decreased markedly even when there was no known increase in the vitamin A in their diet. Of the 25 children giving consistently subnormal or borderline readings, on repeated tests 16 still did not show normal thresholds after intensive vitamin A therapy. They concluded that, owing to the variability in the results of successive tests, the tendency to improve with practice and the uncertainties as to the range of normal readings, the biophotometer does not provide an accurate or a reliable means of detecting mild degrees of vitamin A deficiency in school children.

Isaacs, Jung and Ivy [33] have presented some pertinent evidence as to the reliability of the results obtained with the biophotometer and their validity as a criterion of vitamin A deficiency. They pointed out

TABLE 2.—*Reproducibility of Successive Tests*

Subject	Initial Reading		Reading After Ten Minutes of Dark Adaptation	
	First Test	Second Test	First Test	Second Test
1	0.240	0.240	0.008	0.003
2	0.315	0.216	0.008	0.006
3	0.470	0.260	0.009	0.009

the advantages of plotting the data in terms of the logarithm of light intensity (or in terms of dial readings, which are proportional to log I in this instrument) in order to avoid the artificial crowding together of the high and average readings and the isolation of the low readings. They criticized the prevailing methods of obtaining norms in which "low" readings are assumed without proof to be "subnormal" readings. They showed also that in order to draw the conclusion that improvement in dark adaptation is actually due to vitamin A therapy and not to the normal operation of the laws of probability it is necessary to test both a control group and a treated group of supposedly subnormal subjects.

These authors used the biophotometer for testing 143 medical students, modifying the technic of giving the test so as to avoid the aforementioned fallacies. They found that the dietary intake of vitamin A showed no detectable correlation either with biophotometer readings or with clinical signs and symptoms of vitamin A deficiency.

32. Palmer, C. E., and Blumberg, H.: The Use of a Dark Adaptation Technique (Biophotometer) in the Measurement of Vitamin A Deficiency in Children, Pub. Health Rep. **52**:1403-1418 (Oct.) 1937.

33. Isaacs, B. L.; Jung, F. T., and Ivy, A. C.: Vitamin A Deficiency and Dark Adaptation, J. A. M. A. **111**:777-780 (Aug. 27) 1938.

Friderichsen and Edmund [34] described a method of studying the light sense of young children between 2 and 24 months of age. It was found that a light stimulus produced certain definitely recognizable reflex movements in a child, such as squeezing of the eyelids together and frowning with upward movement of the eyebrows and turning of the eyes or the head toward the light. The source of light used in making the test was standardized photometrically and was checked before and after every experiment. Tscherning glasses inserted in front of the light source were used to vary it by known amounts. The smallest gradation in intensity was 0.25 logarithmic unit, corresponding to a change of approximately 78 per cent. The child was kept in the dark for thirty minutes, and at the end of this period the lamp was caused to emit flashes of from one to two seconds' duration. Ten or twelve flashes were given at each intensity from various positions in the child's field of vision, starting at the lowest intensity and increasing until the child showed a reaction to the light.

It was found possible by this method to make reproducible determinations of the light threshold of babies and to demonstrate a definite and rapid improvement in several with low thresholds, in some instances within one-half hour after the administration of animal or vegetable vitamin A.

Methods of testing the light difference, developed at the University of Copenhagen, have been used by a number of investigators in Denmark and recently by Mutch and Griffith in England.

Edmund and Møller [35] devised test charts, composed of a series of letters, varying from black to a light gray, on a white background. The visual angle subtended by the letters corresponds approximately to 6/36 type on Snellen's charts. Consequently, recognition of the letters depends on the subject's light difference rather than on his visual acuity and is therefore uninfluenced by uncorrected errors of refraction. The faintest letter which can be recognized is taken as the measure of the subject's power of distinction. In the test charts the gradations in reflection power of the letters from black to a light gray are such that the fraction $\frac{\text{difference in reflection power between letter and background}}{\text{reflection power of background}}$ decreases in a geometric progression, namely,

$$\tfrac{1}{10^{0.25}},\ \tfrac{1}{10^{0.50}},\ \tfrac{1}{10^{0.75}},\ \tfrac{1}{10^{1.0}},\ \tfrac{1}{10^{1.25}},\ \tfrac{1}{10^{1.50}},\ \tfrac{1}{10^{1.75}} \text{ and } \tfrac{1}{10^{2.0}}$$

The corresponding values of power of distinction are 0.25, 0.50, 0.75, etc., since power of distinction is defined as $\log \frac{B}{B-T}$, in which B is the reflection power of the background and T that of the test letter. In

34. Friderichsen, C., and Edmund, C.: Studies of Hypovitaminosis A: II. A New Method for Testing the Resorption of Vitamin A from Medicaments, Am. J. Dis. Child. **53**:89-109 (Jan., pt. 1) 1937.

35. Edmund, C., and Møller, H. U.: Vision in Light of Reduced Intensity, Arch. Ophth. **54**:531-544, 1925.

a given individual the power of distinction varies with the intensity of illumination of the test chart. In accordance with Tscherning's suggestions, the illumination is specified on a logarithmic scale. On this scale 5.0 Tscherning units (photopters) are equal to 1 meter candle; consequently, the zero of the scale corresponds to an illumination of 10^{-5} meter candles. In making the tests of the power of distinction at different levels of illumination, the test chart is illuminated by a constant high intensity. Tscherning's photometric glasses are used to reduce the effective illumination on the test chart. For example, if the illumination on the test chart is 10.0 photopters and a glass of density 6.0 is placed in front of the subject's eye, the effective illumination is 4.0 photopters.

A complete test of the power of distinction and its variation with intensity involves preliminary dark adaptation of the eye for one-half hour, followed by determinations of the power of distinction at each illumination, starting with the lowest. With each increase in intensity, sufficient time is allowed for adaptation before any tests are made. At the lowest intensities, according to Edmund and Møller, the results may be influenced by the physiologic central scotoma which occurs in the dark-adapted eye. They suggest that if the patient has difficulty in "finding" the test letters he be allowed to trace them with a pointer. A shortened form of the test is sometimes used, in which the power of distinction is determined only for an illumination of 4.0 photopters (0.1 meter candle). This illumination was chosen because a preliminary adaptation of three minutes or less is sufficient and because it is above the level at which a central scotoma occurs.

Edmund in 1926 [36] examined 30 normal students by means of these tests for power of distinction. He found that at brightness level 4.0 the power of distinction was 1.5 for 27 of the 30 students and slightly lower for the other 3. In a subsequent work in 1932 Edmund [37] measured the power of distinction for a range of intensities from 0.0 to 9.75 photopters. The normal eye showed a constant power of distinction of 2.0 for illuminations between 5.0 and 8.0 photopters. Between 5.0 and 0.0 photopters there was a gradual decrease in power of distinction with a decrease in intensity of illumination. There was also a decrease in power of distinction with an increase in intensity of illumination from 8.0 to 9.75 photopters. Similar tests on 6 patients, 2 with congenital night blindness and 1 each with nutritional night blindness, pigmentary degeneration of the retina, choroiditis and glaucoma, gave lower values for the maximum power of distinction and a reduction in the range of intensity over which the power of distinction was at

36. Edmund, C.: Some Methods of Testing Dark Vision, Acta ophth. **3**:153-169, 1926.

37. Edmund, C.: Dysaptatio Visualis, Tr. Ophth. Soc. U. Kingdom **52**:249-257, 1932.

its highest. Edmund concluded from these findings that there is no true distinction between night blindness and day blindness, since the light difference is affected both at high and at low intensities.

Recent investigations in which the test for power of distinction has been used include those of Frandsen, Brändstedt, Edmund and Clemmesen in Denmark, and Mutch and Griffith, in England.

Frandsen [38] in 1935 reported the results of an extensive study the objects of which were to determine (*a*) whether the power of distinction is subject to individual differences, (*b*) whether it shows spontaneous variations, possibly of a seasonal nature, and (*c*) whether it may be influenced by the administration of vitamin A. A group composed of presumably normal school children and normal adults tested at intensity 4.0 photopters showed powers of distinction ranging from 0.75 to 1.75. Repeated examinations of the same person showed that the power of distinction varied, not from day to day but in the course of some months. In untreated persons the individual values varied from 1.0 to 1.5. Poor power of distinction was found in persons on an institutional diet low in vitamin A. From these findings and from a study of the improvement produced by vitamin A therapy, Frandsen concluded that the normal power of distinction at level 4.0 photopters lies between 1.5 and 2.0. Complete recovery from the defective power of distinction as well as from the concurrent symptom complex was obtained in most cases only by a combined treatment with a preparation of halibut liver oil with added vitamin D (decamin) and calcium phosphate.

Brändstedt [39] in the same year reported statistical studies showing the effect of such factors as age and refractive error on the light minimum and on the power of distinction. At intensity 4.0 photopters the power of distinction was less than 1.25 in 12.7 per cent of nonmyopic eyes and in 34 per cent of myopic eyes. The lowered power of distinction found in myopes occurred in those with normal eyegrounds as well as in those with atrophic changes but was less marked in myopes with normal fundi. Both myopes and nonmyopes showed a definite decrease in power of distinction with increase in age.

Edmund and Clemmesen [40] found that pregnant women with a low power of distinction showed an improvement when given intramuscular injections of a vitamin A preparation.

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38. Frandsen, H.: Hemeralopia as an Early Criterion of A-Avitaminosis and Clinical Symptoms and Treatment of This Disease, Acta ophth., 1935, supp. 4, pp. 1-160.

39. Brändstedt, G.: Untersuchungen über Minimum perceptibile und Distinktionsvermögen des Auges besonders hinsichtlich ihres Verhaltens bei Myopie, Acta ophth., 1935, supp. 5, pp. 1-188.

40. Edmund, C., and Clemmesen, S.: On Parenteral A Vitamin Treatment of Dysaptatio (Nyctalo-Hemeralopia) in Some Pregnant Women. Acta med. Scandinav. **89**:69-92. 1936.

Mutch and Griffith [41] measured the power of distinction at seven levels of illumination between 1,670 meter candles (8.22 photopters) and 1.670 X 10^{-3} meter candles (2.22 photopters). They examined 130 persons and from the results determined limiting values of the normal power of distinction at each intensity. They found on repeated tests of the same person that there was no appreciable improvement with an increase in vitamin A intake in those subjects whose curve of power of distinction was initially within normal limits. Those with initially low values, however, showed improvement after the vitamin A intake was increased. Illustrative data from this report showing (*a*) the upper and lower limits of normal and (*b*) the average data for 3 mill girls who, according to the authors, showed "dramatic improvement" after vitamin A therapy, are given in table 3.

TABLE 3.—*Effect of Vitamin A Therapy on Power of Distinction*

Density of Tscherning glass used	8.00	7.00	6.00	5.00	4.00	3.00	2.00
Log intensity in photopters	0.22	1.22	2.22	3.22	4.22	5.22	6.22
Power of distinction							
(*a*) Upper limit of normal	0.50	0.75	1.00	1.25	1.50	1.75	2.00
Lower limit of normal	0.25	0.50	0.75	1.00	1.25	1.50	1.75
(*b*) Average for 3 mill girls before vitamin A therapy	....	0.18	0.50	0.75	1.16	1.41	1.58
Average after vitamin A therapy	0.00	0.50	0.83	1.16	1.33	1.58	1.83

(Separate data for each of the 3 subjects were not given.) It should be noted that the average improvement in the power of distinction is less than 0.50 at all intensities and therefore less than the normal variation found by Frandsen on repeated tests on the same person.

The most recently described apparatus for the measurement of dark adaptation is that of Hecht and Schlaer.[42] This adaptometer has a uniformly illuminated circular preexposure field about 30 degrees in diameter. The maximum brightness of the preexposure field is about 6,000 millilamberts. Lower brightnesses may be obtained by the use of one or more neutral filters with transmissions of 1/4, 1/10, 1/100, 1/1,000 and 1/10,000. Five sizes of circular test field, ranging from 9.5 degrees to 1 degree, are available. The brightness of the test field is varied by means of a neutral wedge and the aforementioned neutral filters. Color filters transmitting respectively above 680 millimicrons and below 460 millimicrons may be inserted if it is desired to determine the adaptation curve for red and blue light. The time of exposure is regulated by means of a camera shutter. The region of the

41. Mutch, J. R., and Griffith, H. D.: A Study of Diet in Relation to Health: Dark Adaptation as an Index of Adequate Vitamin A Intake; Technique and Preliminary Results, Brit. M. J. **2**:565-570, 1937.

42. Hecht, S., and Schlaer, S.: An Adaptometer for Measuring Human Dark Adaptation, J. Optic. Soc. America **28**:269-275, 1938.

retina tested is controlled by means of a luminous fixation point which can be placed to the left or to the right of the test field at any distance up to 20 degrees from the test spot. No means are provided for standardizing the size of the subject's pupil. Data for this instrument showing the range of variation in the adaptation curves of a group of normal eyes have not yet been published.

COMMENT

1. *Standardization of Variable Factors.*—In the various methods described for the clinical study of dark adaptation, both the pupillary diameter and the level of previous light adaptation were standardized only by Derby and his co-workers, Ferree and his co-workers, Matthey, and Feldman. Accardi found that differences in the level of previous light adaptation had a negligible effect on the thresholds determined after thirty minutes of dark adaptation. Delaney agreed with this view and expressed the belief also that differences in the size of the pupil were of little importance because of daily variation in the sensitivity to light of a normal subject.

The region of the retina tested and the means used to control fixation differed markedly in the different studies. Of those who used some form of fixation target, Matthey and Accardi tested the light sense in a single parafoveal region, Hertel tested the fovea and a perifoveal zone, and Feldman, Cucchia and Zanettin all made tests in a number of different parts of the retina. Hecht's apparatus provides means for testing at any place in the horizontal meridian out to 20 degrees from the fovea on either side. No fixation target was used by Derby and his co-workers, Ferree and his co-workers, Delaney or Spaeth. The large test field (72 degrees) used by Spaeth presumably eliminated the necessity for control of fixation. Derby and his co-workers expressed the belief that their procedure measured the light sense at the macula, except in the case of some uncooperative patients who might take a slightly excentric fixation. They stated that in the dark-adapted eye this variation in direction of fixation does not influence the results. Delaney, on the other hand, assumed that the region tested was parafoveal as a rule but that some uncooperative patients fixated foveally rather than excentrically. Ferree, Rand and Stoll stated that with their tests, because of the size of the test field (3 by 10 degrees), little or no difficulty was experienced in maintaining central fixation. The five point field used with the Birch-Hirschfeld instrument and with the biophotometer may aid in maintaining fixation, so that perhaps the fovea, but more probably a parafoveal, region is tested by these instruments. The test for "power of distinction" devised by Edmund and Møller is in all probability a test of foveal sensitivity at higher intensities and of sensitivity in a parafoveal region at the lower intensities.

2. *Regional Variation in Sensitivity to Light.*—Of the recent investigators, only three, Cucchia, Zanettin and Feldman, have reported comparative studies of the light sense in a number of different retinal regions. Neither Cucchia nor Feldman found any definite evidence of localized disturbances in sensitivity to light. Zanettin, in testing patients with glaucoma, found greater defects in certain regions corresponding to the defects in the visual field. Many of the earlier investigators, however, have emphasized the importance of testing the light sense in different retinal regions.[2] Wölfflin [6] recognized that a true picture of the effect of pathologic conditions on the sensitivity of the dark-adapted eye can be obtained only by testing a number of different retinal regions.

3. *Studies of Light Sense in Different Pathologic Conditions.*—Of the clinical studies of light sense published since 1929, the greater number have dealt with either glaucoma or vitamin A deficiency. Other pathologic conditions have not been studied sufficiently to permit of final conclusions. In spite of the many patients with various stages of glaucoma studied by several different investigators, there is little general agreement as to the clinical value of such tests in diagnosis and prognosis. Those who have studied vitamin A deficiency, however, are in general agreement that a reduced sensitivity to light in scotopic vision is, except in extreme cases, not only the earliest but the only detectable sign of this condition. The different investigators disagree, however, as to the frequency of vitamin A deficiency and as to the reliability of the light sense test in detecting it. Lack of adequate criteria for distinguishing between pathologic and normal sensitivity to light probably accounts for many of the conflicting findings in the various clinical studies.

4. *Range of Variation in Normal Eyes and Determination of Borderline Between Normal and Pathologic.*—Many of the investigators reported data showing the range of variation of the light minimum in a group of subjects without an ocular pathologic process. The results of these different studies cannot be expected to show similar absolute values of the light minimum, since the experimental conditions as to brightness of the preexposure field, size of the test field and region of the retina examined were not the same in the different investigations. With the exception of the data of Matthey and of Jeghers, however, all show surprisingly close agreement in that the greatest value of the light minimum for the supposedly normal group was in every case approximately double the average value. Matthey's data indicate a greater range of variation. The borderline value of the normal light minimum proposed by Matthey was, however, computed from the average normal value from statistical considerations, not determined empirically, as in the other investigations. Jeghers did not present any data on the average value of the light minimum for the group of normal eyes tested with the biophotometer. The threshold values determined

after ten minutes of dark adaptation, however, ranged from a minimum of 0.0148 milli-footcandle to a maximum value approximately 8 times as great. These values indicate a considerably greater range of variation in the light minimum as determined with the biophotometer than was found with the various other instruments and methods of testing.

The problem of determining for a given instrument and technic the borderline between normal and pathologic values of the light minimum presents definite difficulties. It has been generally assumed that a group of subjects without any apparent ocular pathologic process could be used to determine the range of normal variation. The recent studies of vitamin A deficiency indicate, however, that this condition may be of frequent occurrence and may cause a marked reduction in the light sense without any associated objective ocular changes and in many cases without symptoms. If these findings are borne out by further investigation, it is obvious that in obtaining normal limits for a given form of a test for light sense, the group of persons tested must be composed of subjects in whom one can definitely rule out not only an ocular pathologic process but also vitamin A deficiency and any other conditions (such as those suggested by Feldman's work) which may reduce the light sense. Interpretation of the findings is difficult, moreover, even in those cases in which the light sense is obviously reduced. The possible causes for the reduced sensitivity to light which must be kept in mind include various forms of ocular pathologic conditions, vitamin A deficiency, perhaps other general physical ailments and, in addition, such factors as poor cooperation on the part of the subject, inadequate control of fixation and errors in technic.

If further studies of the light sense, for example, comparative studies in different retinal regions, are found to reveal qualitatively different types of defect associated with different causes, as is true to a certain extent in studies of the visual fields, then interpretation of the results of determinations of the light sense may be made easier and the diagnostic and prognostic value of such a test increased. In conditions such as localized choroidal and retinal lesions, or simple glaucoma, for example, one would expect to find regional defects in light sense. On the other hand, vitamin A deficiency, retinal arteriosclerosis, acute glaucoma and any other conditions which probably have an effect throughout the entire retina might cause defects in light sense which were demonstrable in any region of the field. Pathologic changes affecting primarily the rods would be expected to show early defects in scotopic vision, whereas those affecting primarily the cones would be better detected by tests of photopic vision.

It is apparent from this review of the literature that a number of problems require further investigation before tests of light sense will be of much value in clinical ophthalmology. In later papers I shall present data on some of these questions.

ANGIOID STREAKS

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(Concluded from page 774)

THE CHOROID

Besides the pathologic condition of Bruch's layer, that of the choroid attracts immediate attention, even in sections observed under low magnification. A normal choroid, preserved in a solution of formaldehyde, appears collapsed, thin and strongly pigmented, with flat, intensely staining nuclei, so that it requires careful examination to locate the arteries. Occasionally it may be thicker, but in such instances its veins are so filled with blood that it gives the impression of cavernous tissue in which the veins predominate, the arteries being pushed to the background. In my sections the choroid was decidedly thick at the posterior pole, but overfilled veins were not seen, or only exceptionally. Instead, there were many thick-walled arteries present, which in certain areas completely filled the space between Bruch's layer and the sclera. The choroid was moderately and irregularly pigmented and looked rather pale. Sometimes it appeared as though the pigment had been squeezed into the tissue between two arteries. Thus the choroid acquires a typical aspect in which the arteries are the most outstanding feature (figs. 23 *C*, 26, 32 *C* and 40).

Appearance of Arteries in Angioid Streaks and Arterio(lo)sclerosis.—No satisfactory definition of an arteriole exists (Moritz and Oldt [37a] but two varieties of diseases of the choroidal arteries were found. Thickened arteries in association with angioid streaks called for a parallel study of the arteries in arterio(lo)sclerosis of the choroid. The condition of the vessels in arteriosclerosis was studied first:

In eye P. A. 1129 the sclerosis of larger vessels dominated the picture, but its distribution was so irregular that next to a seriously damaged vessel a but slightly sclerotic vessel was found. In the early stages there was already a slight fatty infiltration of the wall of the vessel, but the dominant feature was the proliferation of the cells of the intima. The internal elastic layer, which at first was undulated and apparently normal, gradually became more and more stretched, apparently due to the proliferation of the intima. It coarsened and stained intensely with orcein, so that with this dye the choroid as a whole assumed a typical

37a. Moritz, A. R., and Oldt, M. R.: Arteriolar Sclerosis in Hypertensive and Non-Hypertensive Individuals, Am. J. Path. **13**:679, 1937.

aspect (fig. 23 *A* and *B*). Finally, in the most diseased vessels the stretching of the elastic layer reached its limit, and the original wall of the vessel became thin and atrophic (fig. 21). The proliferation of the intima subsided and showed fatty degeneration, leaving a small lumen surrounded by swollen endothelial cells, which might still permit the passage of blood (fig. 24 *A*).

In eye P. A. 758, from a person with arteriolosclerosis of the choroid, numerous small vessels, arterioli, had thick walls either with many well staining nuclei or partly or completely hyalinized and small or even

Fig. 23.—The sections show the typical aspect of the arteries and choroid in low magnification. *A* and *B*, arteriosclerosis. Depigmented specimen. Orcein stain; × 60. Note the irregular coarse aspect of the choroidal arteries. *C*, angioid streaks. Depigmented specimen. Orcein and hematoxylin stain; × 72.

obliterated lumen. Proliferation of the intima was less evident (fig. 24 *B*). The larger arteries were relatively remarkably well preserved. They showed moderate or no proliferation of the intima; the wall was somewhat vacuolated and thicker than in normal arteries.

In the eye (P. A. 980) of an 82 year old man the arteries in the thin choroid showed no marked changes. In the sections stained with hematoxylin the internal elastic layer of certain vessels stained faintly, so that it was clearly visible.

Another type of degeneration was found in eye Str. 1497, enucleated because of absolute glaucoma. Flattening of the small arteries with hyaline degeneration of the walls probably was the result of the compression (fig. 25).

In eyes with angioid streaks larger arteries were chiefly affected; hyaline degeneration of arterioli, as seen in eye 758, was practically

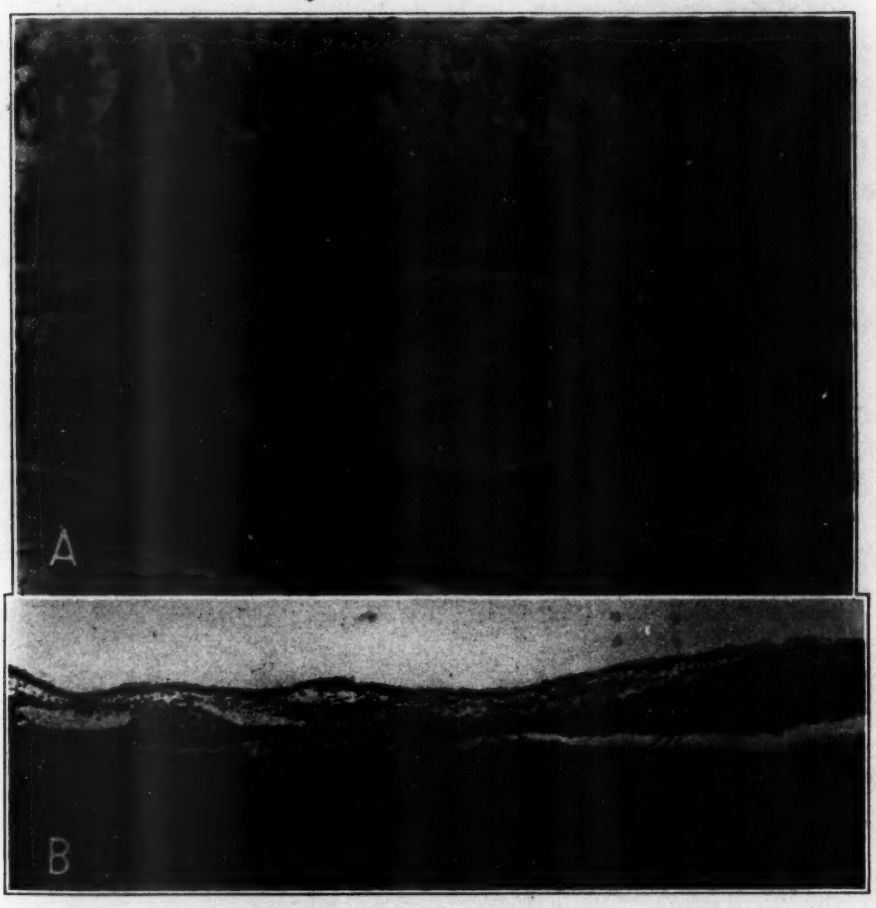

Fig. 24.—*A*, arteriosclerosis. Orcein and hematoxylin stain; × 295. The elastic layer of the artery is distended. There are considerable proliferation and degeneration of the intima, only a small lumen remaining. *B*, arteriolosclerosis of the choroid. Hematoxylin and eosin stain; × 45. Even the smallest vessels are closed by proliferation of the intima. The small vessel second from the right is partly hyalinized.

absent. The involvement of the arteries was rather generalized, being less pronounced toward the periphery. The wall of the vessel was always considerably thickened, so that under low magnification the arteries were

readily seen in the choroid (fig. 23 *C*). This thickening resembled hypertrophy rather than degeneration and was particularly well seen in the sections stained with Van Gieson's stain, the decidedly hypertrophic yellow wall of muscle being surrounded by a stout red wall of connective tissue (figs 26, 31 and 32 *C*). The internal elastic layer was undulated and apparently normal. The intima when not normal showed but slight proliferation. It is interesting to compare these arteries with a large overfilled normal artery of a sarcomatous eye (fig. 27). In cases in which the condition was more advanced the arterial wall was slightly vacuolated, the tissue was less fibrillar, the nuclei stained less and the elastic tissue was atrophied. The internal elastic layer, however, maintained an apparently normal aspect, even when elsewhere all traces of the

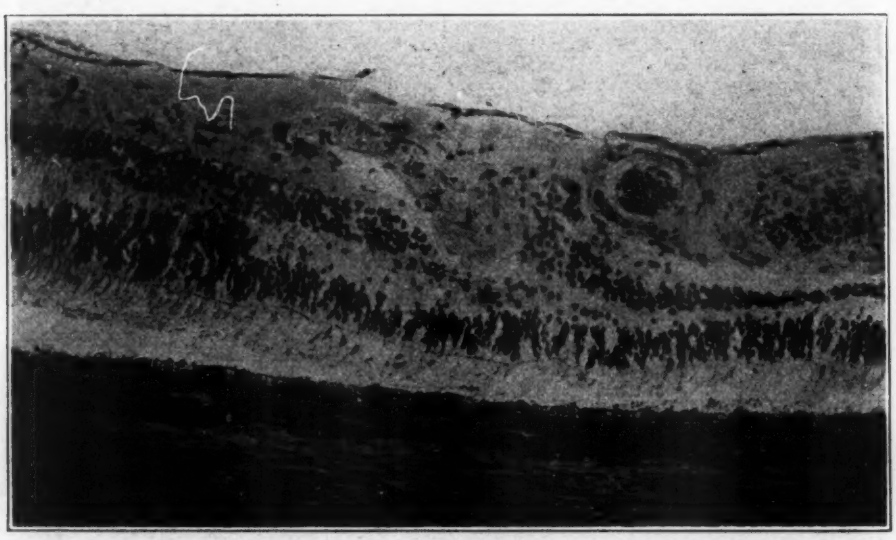

Fig. 25.—Absolute glaucoma, with hyaline degeneration of the flattened arteries. Van Gieson's stain; × 115.

elastic tissue had completely vanished (fig. 28). Occasionally an artery was seen in which this elastic layer finally lost its normal appearance; there were proliferation and degeneration of the intima, and by atrophy of the elastic tissue the arterial wall came into direct contact with the intima (fig. 16 *A*). This maximally diseased vessel did not, however, attain the size of the degenerated arteries in eye P. A. 1129. Though the arteries generally seemed to be hypertrophic rather than degenerated, in some of them the wall was more or less vacuolated, leaving the possibility open of the solution of alcohol-soluble substances. The thickened arteries were often covered by a sheath (external elastic layer), a loose fibrillar and cellular tissue, which was thin between the wall of the vessel and the sclera, Bruch's layer, but well developed on both sides of

the vessel. In this tissue peculiar large cells were seen, which were occasionally encountered elsewhere in the choroid. They are not specific for angioid streaks.

Fig. 26.—Angioid streaks. Van Gieson's stain; × 450. There is a distinct choriocapillary layer. The wall of the artery is thickened. The bright inner layer stained yellow and showed well preserved nuclei. There is an absence of proliferation of the intima.

Fig. 27.—Sarcoma of the choroid. There is an overfilled normal ciliary artery in the neighborhood of the sarcoma. Note the delicate wall of this large vessel.

Appearance of Choroid Outside the Arteries.—The choroidal tissue in eye 980 (82 year old man) was collapsed and densely pigmented; in eye 1129 with arteriosclerosis it was loose (fig. 23 *B*); in the eye with

angioid streaks it was thickened and more solid (fig. 23 *C*), especially at the posterior pole. The nuclei in the first two eyes were thin and deeply stained; in the eye affected with angioid streaks they were oblong and of varied shapes and were paler staining. The veins had disappeared for a great part. Though the choroid might be called sclerotic, still here and there it had a vacuolated appearance. There was only atrophy of vessels, not of the choroid as a whole, since at the most diseased areas it was not thinner but thicker than normal. It is better not to speak of an atrophic but of a sclerotic choroid (fig. 29).

Elastic Tissue in Choroid and Arteries.—In the sections studied the internal elastic layer of the arteries was found to be very resistant. The elastic tissue of the choroid was normal in the periphery but was com-

Fig. 28.—Angioid streaks. Depigmented specimen. Orcein stain; × 850. The choroid is sclerotic. The section shows disappearance of the elastic tissue and preservation of the inner elastic layer of the artery but no proliferation of the intima.

pletely atrophied in some regions at the posterior pole. It stained neither with hematoxylin nor with the Kossa method and was negative to the iron test, thus showing that the disease of the elastic tissue in the choroid is different from that of Bruch's membrane. But in the case of pseudoxanthoma described later in this paper only part of the degenerated elastic fibers of the skin stained with hematoxylin; consequently this peculiar degeneration is not always associated with basophilia. The fibers in the choroid became irregular, as though from loss of tension, but they did not break (fig. 30). They atrophied and gradually disappeared, but did not fall to pieces. In the thick pyroxylin sections they could occasionally be followed over a long distance, running an irregular

course. They were sometimes reduced in size till they were practically no longer visible, but they remained regular. In spite of these general characteristics, there were, however, certain areas in which the degen-

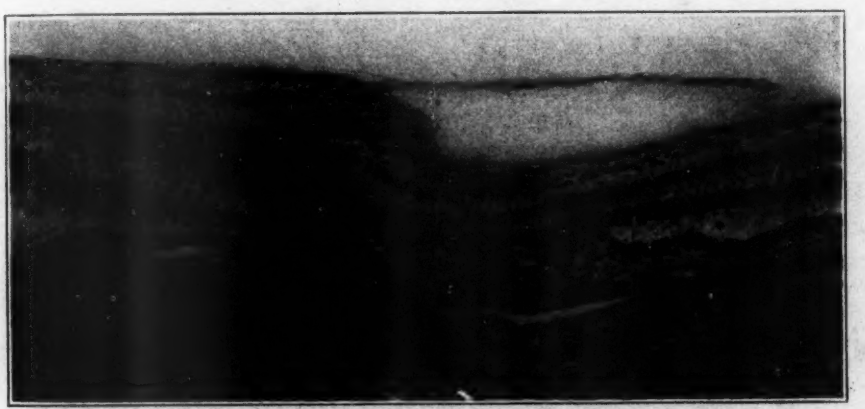

Fig. 29.—Angioid streaks. Hematoxylin and eosin stain; × 75. The choroid is sclerotic; there are ruptures of Bruch's layer and an adherent retina.

Fig. 30.—Angioid streaks. Depigmented specimen. Orcein stain; × 500. The section shows the condition of the elastic tissue of the choroid.

erated elastic fibers were difficult to differentiate from those seen in sections of pseudoxanthomatous skin stained with orcein. In such areas they appeared swollen, were less compact and stained irregularly. The choroid of eye 1129 also (arteriosclerosis) showed degeneration of the

elastic tissue, which in the sections stained with orcein was difficult to differentiate from that seen in angioid streaks. The fibers seemed coarser in eye 1129 and more baked together, the picture of the choroid often being dominated by the coarse aspect of the intensely staining elastic layer of the arteries (fig. 23 *A* and *B*).

It may be concluded that the hypertrophic sclerosis of the choroidal arteries in angioid streaks, though somewhat resembling the condition in the larger arteries in arteriolosclerosis, is nevertheless a different sort of disease. The choroid is sclerotic in the posterior part of the eye. Here the elastic tissue has atrophied; its specific nature, though not improbable, could not be demonstrated with certainty.

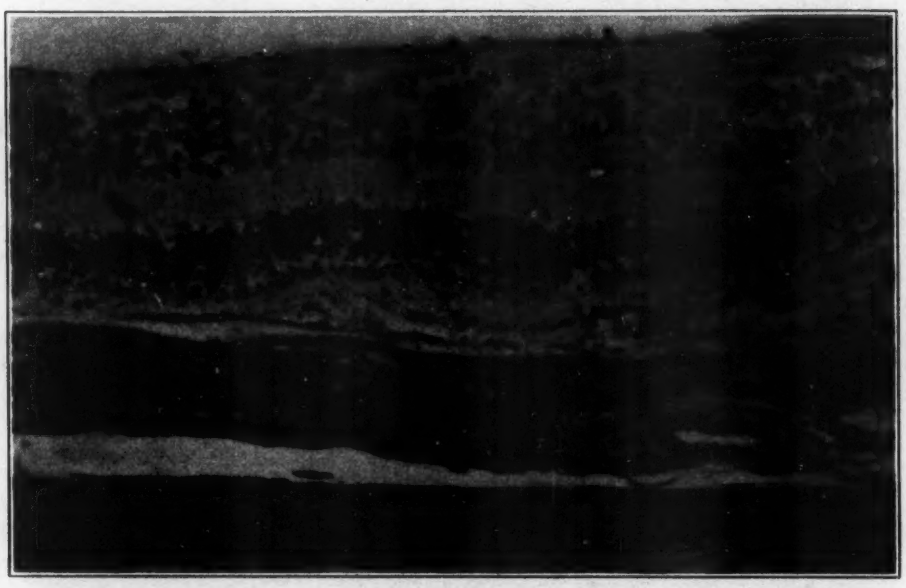

Fig. 31.—Angioid streaks. Macular region. Hematoxylin stain; × 145. The ganglion cells are well preserved. The section shows atrophy of the inner layers of the retina, absence of pigmented epithelium, heaps of lymphocytes in the choroid and a hypertrophic artery with a strong muscular wall but no proliferation of the intima.

Pseudoinflammatory Conditions.—The nuclei in the choroid seemed to be more polymorphic than normal. A considerable number of lymphocytes and an occasional leukocyte were found free in the choroidal tissue. They sometimes even lay in heaps, reminding one of an incipient case of sympathetic ophthalmia (fig. 31). Lymphocytes in the choroid are, however, found under various conditions, trauma, general infections and arteriosclerosis, and without any known cause. Their presence, therefore, does not allow the conclusion that there is an inflammation of chronic character; they may represent the reaction to the pathologic condition of the choroid.

The Pigment of the Choroid.—The pigmented cells were often stretched, showing large protoplasmic cell bodies in which the isolated pigment granules could be readily seen. In the most affected areas the distribution of pigment was irregular; it was practically absent in one area, close to which a number of chromatophores had fused, forming a dense dark mass. In the less affected regions the distribution was regular, the pigment being chiefly present in the outer layers.

The choriocapillaris, though missing over considerable areas, nevertheless showed decidedly more resistance than the veins, which for a great part had completely disappeared. It was sometimes present in seriously damaged regions.

The Pigmented Epithelium.—At the extreme periphery the pigmented epithelium was normal. Connective tissue could be seen between the pigmented epithelium and the retina, as has been recently extensively described by Reichling [38] and previously mentioned by Verhoeff.

At the periphery the pigmented epithelium, as has been described, was pushed off from Bruch's layer, but it appeared to be normal in all other respects. A little further it was seen lying directly on Bruch's layer (fig. 17 *C*). As shown by the paraffin sections, it was pathologic here and fixed to Bruch's layer. In the section shown in figure 32 *A* it had been torn off artificially, leaving a protoplasmic mass attached to the layer. Farther from the periphery the pigmented epithelium was seen to be seriously affected. "Serous exudates" (fig. 32 *B*) in which destroyed pigmented epithelium was scattered were sometimes present. It was difficult to judge whether they were (partly) due to artefact. Toward the posterior pole the pigmented epithelium varied; it was absent, proliferated, formed a homogeneous mass or became apparently fibrillar tissue. Over a large area at the nasal side, the retina though artificially detached, had in vivo grown together with the pigmented epithelium. The detachment ran through the irregularly proliferated pigmented epithelium, so that considerable rests of these cells remained attached on the one side to the other side to Bruch's membrane.

At the posterior pole the pigmented cells sometimes fused, forming dark pigmented heaps, or produced basophilic homogeneous bodies (drusen), which attained a considerable size and were also seen at the periphery. Generally, however, the pigmented epithelium tended to atrophy. Occasionally a small druse was seen projecting toward the choroid, which probably developed independent of the pigmented epithelium.

38. Reichling, W.: Ueber eine Gefässführende Bindegewebsschicht zwischen Pigmentepithel der Retina und Lamina vitrea, Klin. Monatsbl. f. Augenh. **99**: 543, 1937.

THE RETINA

At the periphery the retina was fairly normal, though the rods and cones were as a rule difficult to trace. It was not uncommon, however, even in an eye fixed immediately after enucleation, to find this vulnerable layer damaged and indistinct. At some distance from the disciform degeneration, the retina, which was detached at the periphery, was seen

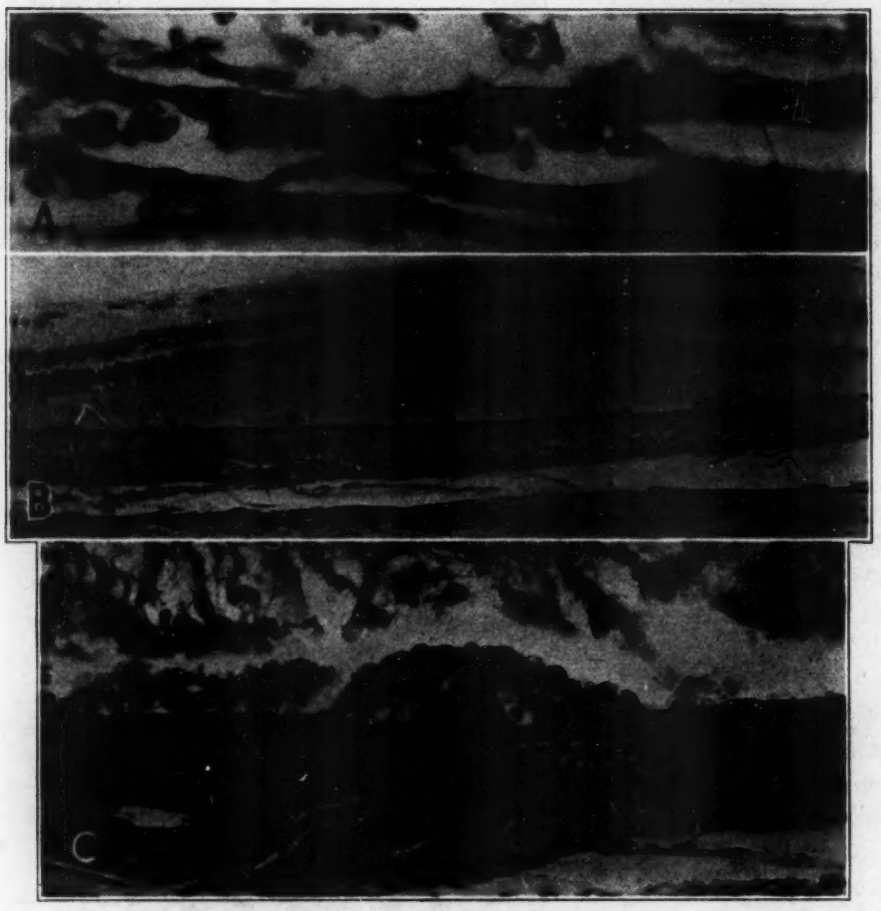

Fig. 32.—*A*, angioid streaks. Orcein (elastic tissue) stain; × 1,050. Bruch's layer is broken, and adherent pigmented epithelium is torn off, leaving a homogeneous layer. Note the connection of choroidal elastic fibers with Bruch's layer. *B*, angioid streaks. Hematoxylin and eosin stain; × 82. A large serous subretinal exudate is present. *C*, angioid streaks. Hematoxylin and eosin stain; × 225. The retina is torn off. There are a homogeneous layer under the pigmented epithelium and hypertrophic arteries with a strong muscular wall but no proliferation of the intima.

fixed to the pigmented epithelium. The rods and cones had completely disappeared, and the outer nuclear layer was seriously affected (fig. 33 *A*). The anterior layers, however, remained normal, leaving no doubt

Fig. 33.—*A*, angioid streaks. Hematoxylin and eosin stain; × 90. In approaching the macular region, the outer nuclear layer disappears and the retina becomes adherent to the underlying tissue. The pigmented epithelium is damaged, is partly missing and is baked together at the right. A thick, intensely stained Bruch's layer, hypertrophic vessels in the choroid and lymphocytic exudates in the choroid are shown. *B*, angioid streaks. Macular region. Hematoxylin and eosin stain; × 80. The ganglion cells of the retina are well preserved. The inner nuclear layer is missing for the greater part. Newly formed tissue with capillaries on Bruch's layer, hypertrophic arteries and dense pigmentation of the choroid are shown. *C*, angioid streaks. Hematoxylin and eosin stain; × 55. There are retinal folds in the macular region.

therefore that the involvement of the retina was secondary to the involvement of the choroid, Bruch's membrane and the pigmented epithelium. Even in the region closer to the macula, where the pigmented epithelium became scattered and newly formed tissue developed under the retina (fig. 33 *B*), it generally maintained the structure of its anterior layers. Only in the most altered regions did the inner nuclear

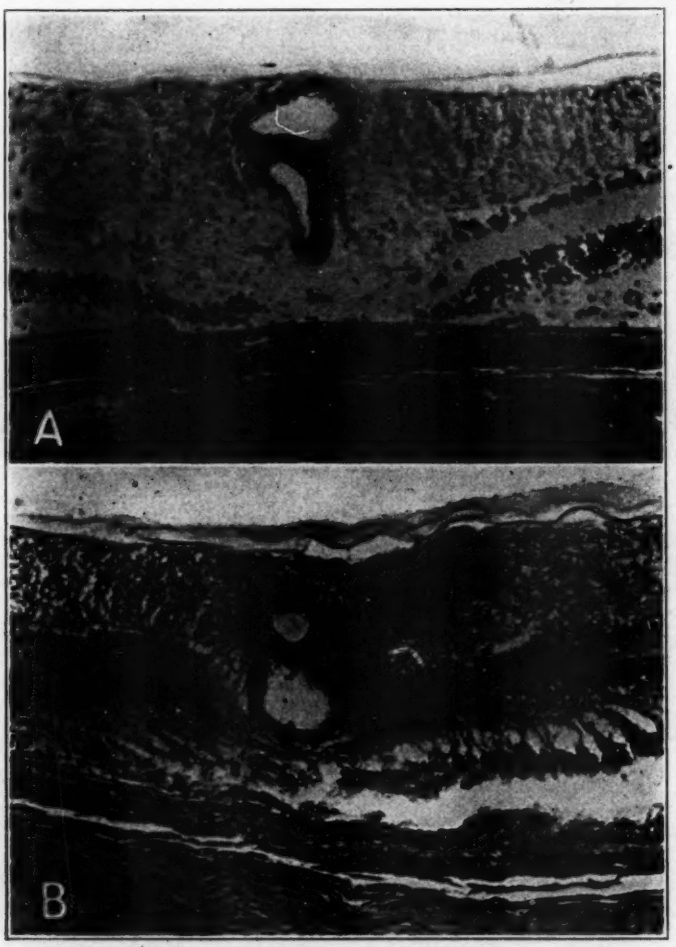

Fig. 34.—Angioid streaks. Retinal vessels. *A*, Van Gieson's stain; × 105. *B*, hematoxylin stain; × 105.

layer become irregular, where for short distances it may even have disappeared completely. The ganglion cells, so far as could be judged from the pyroxylin sections, always remained normal. In the anterior layers of the retina neither holes nor amorphic bodies were found, as is the case in albuminuric retinitis. In the macular region the retina, though folded (fig. 33 *C*), was still attached to the underlying tissue. It

is evident that these folds, which also occurred in Law's case (fig. 6), are not identical with angioid streaks.

Little can be said about the vessels of the retina. Their walls appeared to be thickened, sclerotic, so that in various places the retinal tissue was pushed aside (fig. 34). This is, however, also seen in perfectly normal eyes. Proliferation of the intima was either absent or slight.

The optic nerve did not show a degeneration of fibers with the myelin stain (Kulschitzky).

Newly Formed Tissue Between the Retina and Bruch's Layer.—There was no doubt that a proliferation of glial tissue had occurred.

Fig. 35.—Angioid streaks. Hematoxylin and eosin stain; × 200. There are newly formed loose tissue between the retina and Bruch's layer, probably of a glial nature, and lymphocytes in the choroid.

This loose fine fibrillar tissue (fig. 35) closely resembled that found in a previously reported case of traumatic rupture of the pigmented epithelium (Hagedoorn [39]).

The choroid is surprisingly inactive in closing the ruptures of Bruch's layer. Cicatricial tissue is formed in traumatic ruptures and also in the tears due to myopia. The choroid, however, shows no reaction to a great number of the tears which occur in angioid streaks. In this study

39. Hagedoorn, A.: Choroidal Tears, Am. J. Ophth. **20**:1, 1937.

some of the tears may have resulted from trauma produced in making the sections, but undoubtedly a great number must have been present in the living eye. The inactivity of the choroid accounts for the "angioid" character of many angioid streaks, whereas in myopia the breaks are more often white, owing to the development of this cicatricial tissue.

Nevertheless, in the center of degeneration at the posterior pole and around the disk hypertrophy of dense fibrillar tissue and small vessels was present, which originated from the choroid or from mesodermal elements. It is interesting to note that in the newly formed tissue anterior to Bruch's layer elastic fibers may be present. Consequently, the conditions in the human body under which the elastic tissue developed must have been undisturbed. In this same tissue even a piece of bone had developed (fig. 36 *A*). It was negative to the Kossa test, but normal bone corpuscules could be demonstrated (fig. 36 *B*). Evidently bone had developed without a preceding calcification, or this small exposed piece may have been decalcified by the (acid) solution of formaldehyde. Bone nearly always contains calcium. Schaffer [40] described newly formed bone in the skin of the head which did not contain calcium, the calcium being dissolved by fixation in Zenker's fluid for twenty-four hours. Mebius [41] found metaplastic bone in lung which stained with fuchsin, so that it could contain only a small amount of calcium. Schupisser mentioned a paper by Schmorl, in which the development of bone without calcium was described as being detectable in the first stage. According to Welcker,[41a] bone develops if newly formed (or transplanted) connective tissue touches epithelium.

The pigmented epithelium may form a structureless, nonbasophilic substance and probably also a fibrillar tissue. The latter feature of the pigmented epithelium has been accepted by Rintelen in his case of disciform degeneration. It is a phenomenon which takes its place among other peculiar features of the eye and its cells: embryologically, with the development of muscle tissue from the ectoderm; anatomically, with the structure of the vitreous body; physiologically, with the marked double innervation, the peculiar mode of contraction of the muscles and the interesting metabolism of the lens; pathologically, the development of fibrillar tissue from lens and ciliary epithelium, and oncologically, with the ocular glioma of early childhood. All these pecu-

40. Schaffer, J.: Ueber einen Befund von Knochengewebe in der Kopfhaut beim Menschen, Centralbl. f. allg. Path. u. path. Anat. **18**:721, 1907.

41. Mebius, J.: Over heterope beenvorming naar aanleiding van die in buiklitteekens, Amsterdam, J. H. de Bussy, 1918.

41a. Welcker: Experimentelle Erzeugung heterotopen Knochens, Zentralbl. f. Chir. **65**:2780, 1938.

liarities of the eye point to the fact that it is an organ somewhat apart, an explanation for which can be found in a study on its comparative anatomy (Hagedoorn [42]).

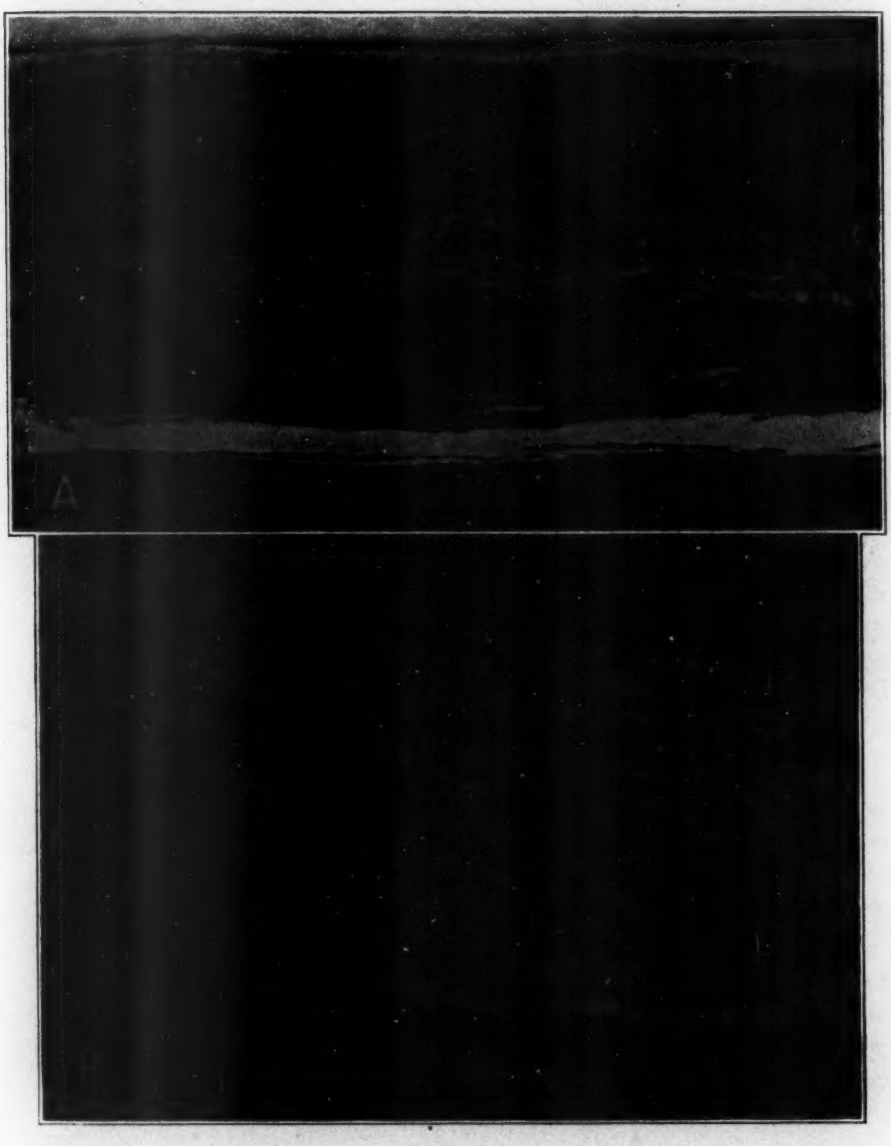

Fig. 36.—*A*, angioid streaks. Hematoxylin and eosin stain; × 130. The ganglion cell layer is well preserved. The outer nuclear layer is missing. Newly formed tissue and bone tissue are shown. Bruch's layer is ruptured. *B*, angioid streaks. Bone corpuscles stain; × 475. Typical bone corpuscles are seen in metaplastic bone.

42. Hagedoorn, A.: Comparative Anatomy of the Eye, Arch. Ophth. **16**:783 (Nov.) 1936.

Peripheral Plaque.—At the nasal side of eye 1335a there was a sharply demarcated region of extreme atrophy of the retina. A reconstruction was made (fig. 37). The atrophy resembled that seen in the the eye of an ape in which a ciliary artery had accidentally been cut in making a lesion in the macular region. A similar atrophy was seen in an eye enucleated because of a herpetic condition with involvement of the choroidal vessels. Wagenmann [43] described similar lesions following closure of ciliary arteries. No definite lesion of a larger ciliary artery could, however, be found. The cause of this degeneration remained a puzzle. In the most damaged regions the retina was reduced to a thin sheet of glial tissue with a few scattered cells (fig. 38). In the less affected areas, the anterior layers were preserved. It is probable, therefore, that this condition of the retina is secondary to a disease of the underlying tissue. Such complete degeneration as was seen in the peripheral plaque was not found at the posterior pole, which was

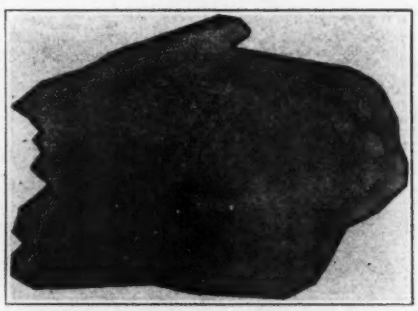

Fig. 37.—Reconstruction of the peripheral plaque.

probably due to the fact that the blood supply of the anterior layers of the retina from the retinal vessels was less perfect in this region than it was at the posterior pole.

Bruch's layer was carefully studied and compared with the same region on the temporal side, but no differences could be traced. The entire nasal side of the choroid seemed to be somewhat thicker, but little value can be placed on this observation. With the van Gieson stain, however, a number of sections showed a group of thick-walled arteries with open lumens lying close together under the affected retina (fig. 39). In the spaces between them had crowded the pigmented cells of the choroid, which gave to the choroid in the sections stained with hematoxylin a peculiar mottled appearance (fig. 40) entirely different from that of the same region on the temporal side. In this same

43. Wagenmann, A.: Experimentelle Untersuchungen über den Einfluss der Zirkulation, Arch. f. Ophth. **36**:1, 1890.

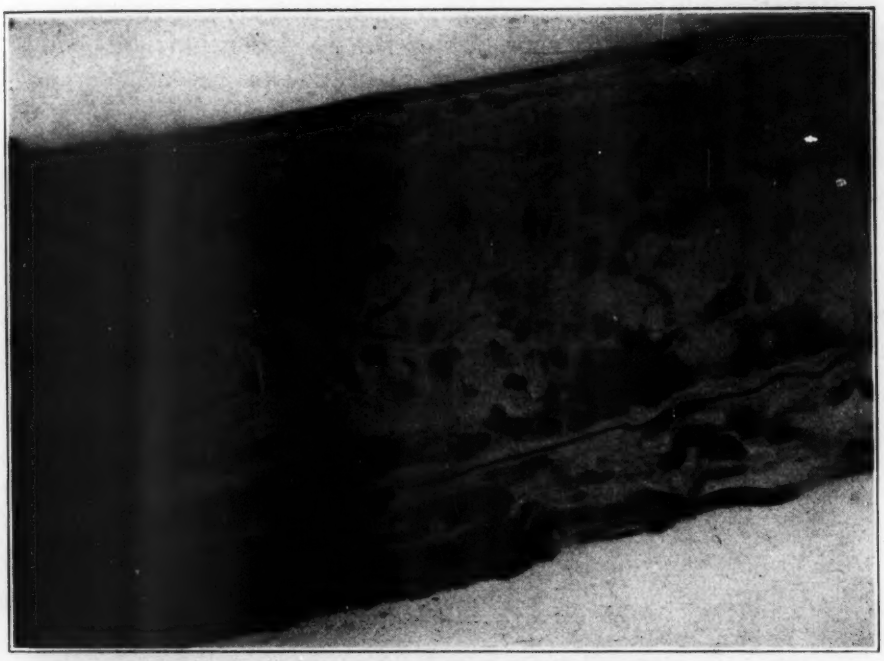

Fig. 38.—Angioid streaks. Degeneration in the peripheral plaque and extreme atrophy of the retina. Hematoxylin and eosin stain; × 540. Bruch's layer is fairly normal.

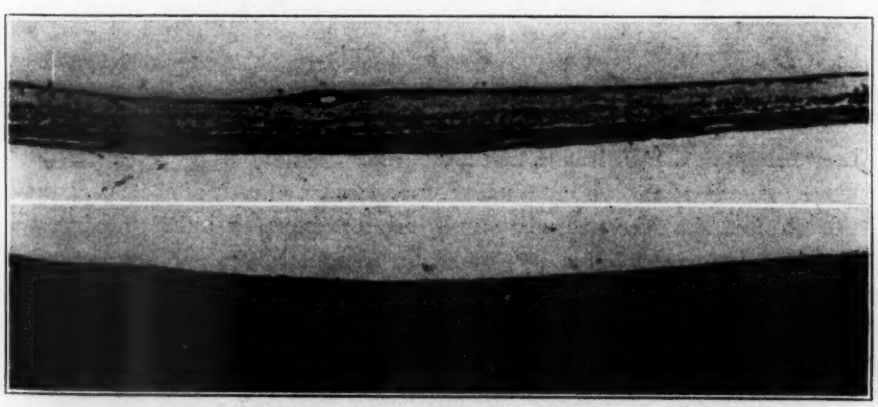

Fig. 39.—Angioid streaks. Van Gieson's stain; × 60. On the nasal side (upper figure) there are open thick-walled vessels and the retina is adherent. On the temporal side no individual vessels are visible and the retina is detached.

region a small, fainter staining area of the choroid was seen, which was devoid of nuclei, evidence that complete degeneration had occurred. Though the condition of the peripheral plaque seems to be a plea in favor of a specific disease of the choroidal arteries, its similarity to the findings in occlusions and diseases of ciliary arteries warns against too absolute a standpoint.

OTHER PARTS OF THE EYE

Sclera and Cornea.—In the sclera the elastic tissue was excellently preserved throughout, and no foci of degeneration of the elastic tissue could be found. The cornea also appeared to be normal. However, in a series of sections of eye 1335a a subepithelial degeneration was found in the region of the limbus. It seemed to be a degeneration of the peripheral part of Bowman's membrane, but this could not be proved,

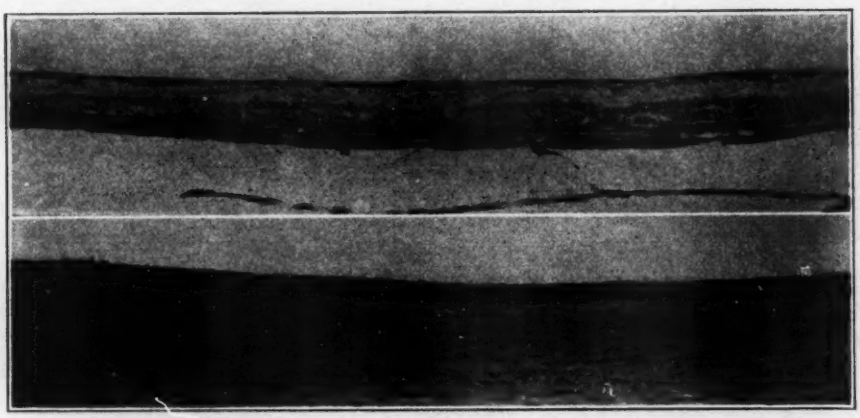

Fig. 40.—Angioid streaks. Peripheral plaque. Hematoxylin and eosin stain; × 60. On the nasal side (upper figure) the pigmented cells of the choroid are pushed aside by thick arteries. On the temporal side the regular distribution of choroidal pigmentation is seen. The retina is detached.

since Bowman's membrane could be only incompletely traced in the cornea. In the same sections a large pinguecula was found. Other eyes were studied for the presence of a similar subepithelial degeneration, but none was found. It appeared as an irregular, amorphous, granular mass, staining intensely with hematoxylin. It may be related with simple band-shaped opacity. Peppmüller [43a] and von der Heydt [43b] described a similar degeneration of the cornea in the level of Bowman's

43a. Peppmüller, F.: Ueber eine bisher nicht beschriebene beiderseits symmetrisch gelegene subepitheliale Hornhauttrübung bei einem Fall von Pagetscher Ostitis deformans, Klin. Monatsbl. f. Augenh. **98**:1, 1937.

43b. von der Heydt, R.: Osteitis Deformans with Pigmented Corneal Degeneration, Am. J. Ophth. **20**:1139, 1937.

membrane in osteitis deformans. The Kossa reaction could not be tried, since all of the sections showing the degeneration had been previously stained.

However, it again attracted attention in the sections tested for the presence of iron. Here it stained an intense blue. Sections showing the degeneration in its full development were not available, but a section through the periphery of the degeneration was highly positive. In addition, the basal layer, and at times even more layers of the corneal epithelium, stained more or less intense blue.

Other eyes were stained for iron, and the corneal epithelium was studied. In the limbic region of eye 980, that of an old man, a scarcely

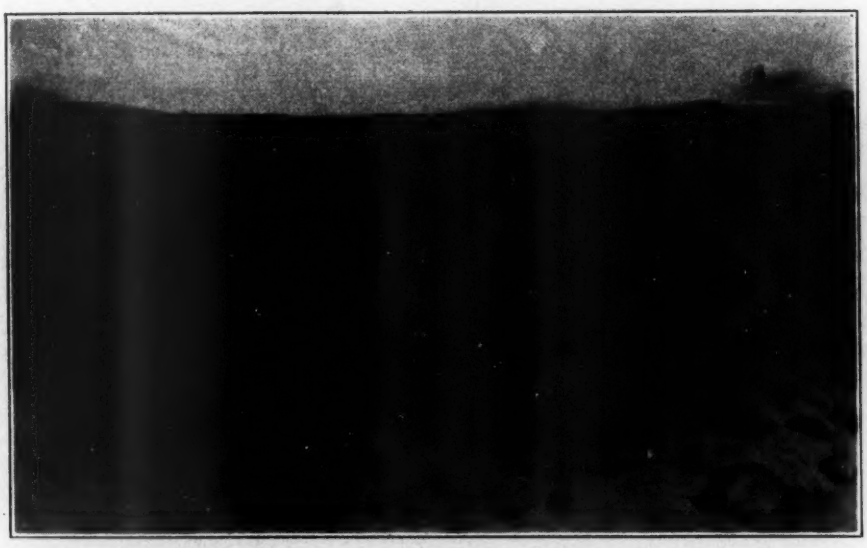

Fig. 41.—Angioid streaks. Section of cornea. Hematoxylin and eosin stain; × 500. There is degeneration of the subepithelial layer (also Bowman's membrane [?]) in the limbic region.

detectable, faint blue tint limited to the basal cells of the corneal epithelium was present. Bowman's membrane was normal. In eye Str. 1338 (iron in the eye) the siderosis was too widespread for minute study, the entire anterior part of the cornea, Bowman's membrane included, staining blue.

In eye 1773 with general siderosis bulbi the basal cells of the corneal epithelium in the limbic region stained faintly. It was, however, so slightly positive that it cannot be considered a proof of predilection for this area, other regions staining much more intensely.

The corneal epithelium in eye Str. 1117, a normal eye with hemorrhage, remained unstained.

It may be concluded that the basal cells of the cornea in the limbic region of older eyes stain faintly with the iron test; a similar staining was present in an eye with siderosis. The staining in these eyes was not, however, so intense as it was in the eye with angioid streaks.

Conjunctiva.—What elastic tissue of the conjunctiva could be studied was fairly well preserved, but in the enucleation the conjunctival folds, which are especially subject to mechanical influences in the movements of the eyes, were cut away. An uncommonly large pinguecula was found. The degeneration of the elastic tissue of the pinguecula differed from that seen in the angioid streaks; it was more dustlike.

Iris, Ciliary Body and Optic Nerve.—These did not show any anomalies.

Ciliary Vessels in the Neighborhood of the Optic Nerve Outside the Eye.—Some of the ciliary vessels in the sections stained with hematoxylin attracted immediate attention, even under low magnification, because of their thickened walls and the unusual visibility of the elastic layer. The elastic layer stained faintly with hematoxylin, whereas in sections of other eyes stained with hematoxylin it remained unstained and could be located only by bringing it in and out of focus, when it was seen as a homogeneous unstained membrane. This unusual visibility, though less pronounced, was also found in some of the arteries of the choroid. It must be emphasized, however, that the staining was faint and could in no way be compared with that of Bruch's layer. In the arteriosclerotic eyes the elastic layer was seen with difficulty. In the eye of an old man, however, a faint staining of the elastic layer was seen, less pronounced than, but similar to, that seen in eyes with angioid streaks. The sections stained with orcein revealed an absence of the elastic tissue of the inner part of the wall of the artery, so that there was a characteristic difference with arteries of arteriosclerotic eyes, studied for comparison (fig. 42 [44]).

COMMENT

Fundus and Histologic Picture.—According to Grönblad, a specific and frequent feature of the fundus in angioid streaks is its spotted appearance. The spots, or dots, may form a mozaic (Wildi [45]) or be irregular (Oeller [46]). In Walser's [47] case the spots were yellowish red,

44. In Böck's case disease of the ciliary vessels could be demonstrated with certainty.

45. Wildi, G.: Zur Fundusentartung mit angioider Streifenbildung, Klin. Monatsbl. f. Augenh. **92**:538, 1937.

46. Oeller: Atlas seltener ophthalmoskopischer Befunde: Zugleich Ergängzungstafeln zu dem Atlas der Ophthalmoskopie, ed. 2, Wiesbaden, J. F. Bergmann, 1903, plate 6.

47. Walser, B.: Drei Fälle eigenthümlicher streifiger Pigmentierung des Fundus, Arch. f. Augenh. **31**:345, 1895.

of the size of a vein of the retina. They increase as the disk is approached; at the posterior pole they form an irregular network. Hartung [48] described a case with a tapetum-like discoloration of the fundus. A similar case is mentioned by Lindner.[49]

Zeeman [50] described a patient with typical pseudoxanthoma elasticum and typical angioid streaks. The diagnosis of pseudoxanthoma was confirmed histologically (Dr. Carol). Examination of a brother and

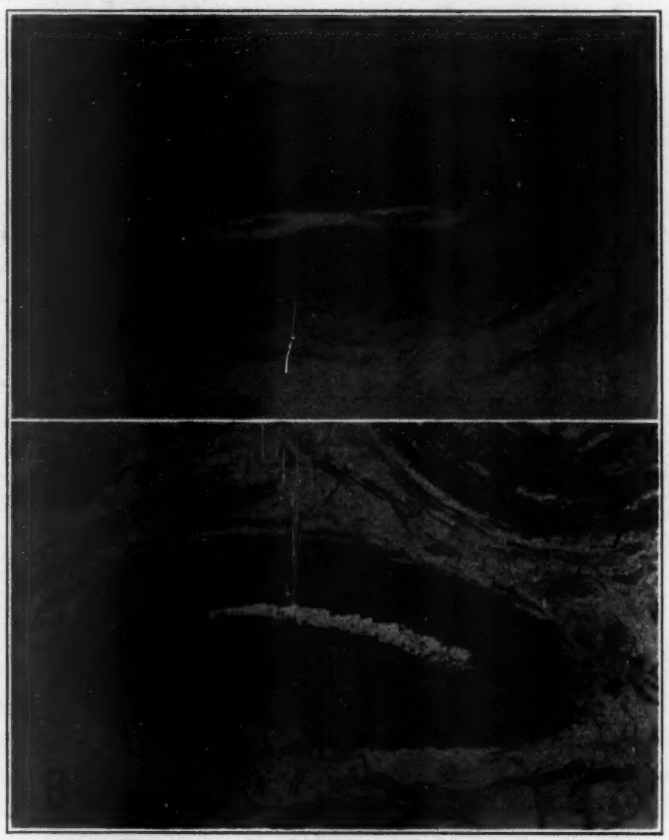

Fig. 42.—*A*, angioid streaks. Ciliary artery. Orcein stain; × 130. *B*, arteriosclerosis. The ciliary artery is of similar size. Orcein stain; × 130.

sister of the patient showed normal fundi at the periphery, but as the center was approached gray dots appeared, which fused at the posterior

48. Hartung, H.: Ueber familiäre angioide Pigmentstreifenbildung des Augenhintergrundes, Klin. Monatsbl. f. Augenh. **88**:43, 1932.

49. Lindner, K.: Ueber Pigmentstreifenbildung in der Retina, Arch. f. Ophth. **88**:230, 1914.

50. Zeeman, W. P. C.: Angioide strepen in de retina zonder angioide strepen, Nederl. tijdschr. v. geneesk. **77**:1939, 1933.

pole, giving to this region a diffuse grayness in which the normal remaining areas could be seen as small red dots.

It is probable that the spots seen by Wildi, Oeller and Walser and Zeeman clinically represent the foci of degeneration of Bruch's layer seen histologically (figs. 13, 14, 17 and 18). The diffuse grayness in Zeeman's case indicated that the foci of degeneration of Bruch's layer have fused and that the membrane in this area has degenerated in toto. Evidently the regions staining so intensely with hematoxylin (as drusen also do) are less transparent in vivo than a normal membrane. When the membrane posterior to the equator is completely degenerated, it becomes so brittle that ruptures can occur spontaneously or following insignificant trauma. The periphery is dotted, as described by Grönblad, the dots being the small areas of degeneration.

Angioid Streaks.—From the foregoing description it may be concluded that if angioid streaks are seen, the membrane in which they have developed is abnormal, its translucency being impaired. Such a membrane acts as a grayish veil, dimming the red color of the choroid (fig. 2); ruptures in it expose the choroid, which accounts for the red appearance of the streaks, their "angioid" character. This explains the visibility of an angioid streak, which would be difficult to understand if the rupture occurred in a membrane the translucency of which was normal. A similar contrast is present in the "red hole" of a detached gray retina.[51]

It is evident that the pigmented epithelium of the retina must play a role in the clinical appearance of the streaks. If the fuchsin bodies, which have a brown-black color, are very numerous in the epithelium they may hide the choroid completely, as a result of which involvement of Bruch's layer will be difficult to observe and those ruptures bridged by intact pigmented epithelium may even escape attention. On the other hand, the pigmentation of the epithelium can be so slight that the fuchsin bodies have but little influence on the red color that the vessels of the choroid give to the streaks. In such eyes they appear as fine brown-black dust. It is in these eyes that the angioid streaks appear as true vessel-like streaks. The color of the angioid streaks depends further on the pigmentation and condition of the choroid. It is probable that the pigmented epithelium ruptures at times along with Bruch's layer, since it is so closely connected with this layer, as has been demonstrated histologically. The pigmented epithelium of a normal eye is vulnerable; it can rupture as the result of blunt trauma (Hagedoorn), the layer of Bruch remaining unimpaired, and in the process of handling the specimens it can break, producing defects resembling angioid streaks (fig. 43).

51. Böck failed to recognize the existence of the veil caused by a degenerated membrane and therefore stated that a rupture in Bruch's layer alone is invisible.

From the histologic picture it is evident that the choroid shows a remarkable inactivity in producing scar tissue. This accounts for the rarity with which angioid streaks are reported as being white in contradistinction to the findings in myopia and trauma, in which scar tissue readily develops.

Hemorrhages.—Hemorrhage in the vitreous body of an eye following trauma is an indication for examining the other eye for the presence of angioid streaks and the skin for pseudoxanthoma elasticum, since angioid streaks have been detected in this way (fig. 7).

Fig. 43.—Sarcoma. Slightly oblique, nearly parallel section through the normal part of the choroid and pigmented epithelium. Orcein stain; depigmented specimen; × 225. To the left is shown pigmented epithelium with artificial ruptures; in the middle, elastic fibers of a normal Bruch's layer; to the right, coarser fibers of choroid.

Though the tendency to hemorrhage in eyes with angioid streaks is decidedly higher than in normal eyes and the occurrence of hemorrhage is often reported in eyes which are carefully studied, the frequent absence, at least of large hemorrhages, is striking. It will be remembered that the basophilia of the elastic fibers of Bruch's layer practically stops as the fiber enters the choriocapillaris. Toward the posterior pole, where the degeneration is more advanced, the layer acquires the aspect of a true membrane which has lost connection with the choroid. It is

obvious, therefore, that a rupture in this membrane may occur without injury to the choriocapillaris, which, moreover, is often obliterated. Night blindness in cases of angioid streaks has been reported in the literature. Anomalies of the color sense may be expected to occur.

Varieties of Involvement of Bruch's Layer in Angioid Streaks.—It has been mentioned that the dots seen in the fundus due to degeneration of Bruch's layer vary in appearance; the ophthalmoscopic picture can be so unusual that their relation to angioid streaks may be easily missed. Such a case was reported by Zeeman in 1933 (fig. 44). The dots were so large that they resembled drusen or a special variety of tapetoretinal degeneration (compare with fig. 14). In the other eye a peculiar angioid streak-like arrangement of dots was seen, because of which the patient was examined for the presence of pseudoxanthoma elasticum. Indeed, a

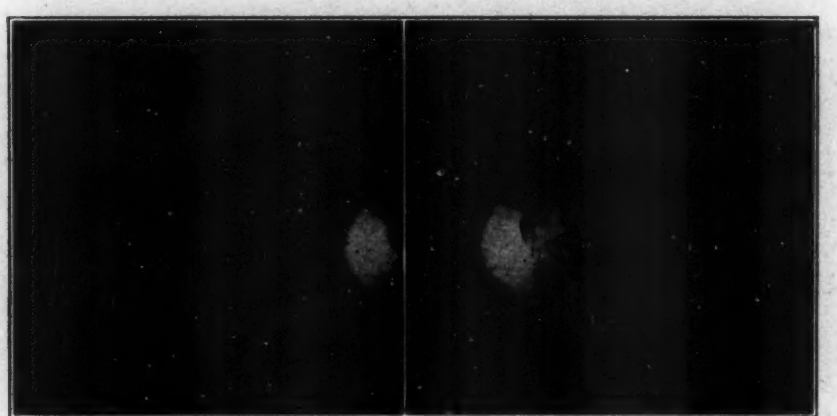

Fig. 44.—Elastic pseudoxanthoma without angioid streaks (Zeeman). Probably the first stage of degeneration of Bruch's layer is shown. (Compare with figure 14). Angioid streak-like arrangement to the right. There are no ordinary angioid streaks.

pseudoxanthoma was present, the diagnosis later being confirmed histologically. The arrangement of the dots in this case suggested the possibility of their being a forerunner of angioid streaks which, had they been developed, would have resembled the condition in those eyes described clinically as having two white lines paralleling the ruptures.

It is interesting to report that a second patient presented a similar fundus, but the presence of angioid streaks could not be established since the dermatologist could neither deny nor affirm the diagnosis of pseudoxanthoma, which also might have been a senile degeneration of the skin. Of further interest in this case was the presence of a marked hypercholesteremia, a disease which has been mentioned by Franceschetti as being associated with angioid streaks.

Fundus and Choroid in Angioid Streaks.—If clinically the circumpapillary and the macular region show white and gray areas, these should not be called atrophic but sclerotic, since from the histologic study it has been seen that atrophy of the choroid nowhere occurred (fig. 29). Clinically, the condition of the arteries is different from that seen in cases of areterio(lo)sclerosis of the choroid. In general hypertension there exists a condition of the choroid, at times described as albuminuric choroiditis (Fuchs [52]), in which pigmented patches indicate the presence of a serious condition of the choroid (Elschnig and Siegrist). These pigmented patches occur as dots and streaks and are often more numerous in the peripheral and equatorial regions than at the posterior pole. A similar lesion of the choroid occurred in an eye unavoidably exposed to radium. This variety of vascular involvement has not been reported in cases of angioid streaks, which is in accordance with their unfavorable prognostic significance, since the prognosis of the hypertension in angioid streaks is good (quoad vitam).

Another variety of arteriosclerosis has been described by Gilbert [53] in the "Kurzes Handbuch der Ophtholmologie"; it usually appears in the earlier stages of general vascular disease and is rarely the indication of a serious general condition. Haab [54] mentioned an earlier stage, with defects in the pigmented epithelium. It has a predilection for the region surrounding the optic disk, more especially the posterior pole. Islands of localized atrophy of choroidal vessels, resembling peripheral plaques, may occur. Though these are undeniable similarities to angioid streaks, still there exist differences: The sclerotic vessels are seen as individual white lines, which at times may still have a small red line in the center. The intermediate tissue between is atrophic only in advanced stages. This variety of arteriosclerosis is at times seen in elderly persons; the circumpapillary atrophy, which may follow, is known as halo senilis. Probably another variety of halo senilis may be associated with the circumpapillary degeneration of Bruch's layer, found in senility. In discussing angioid streaks observers do not mention arteriosclerosis but "atrophy," probably owing to the fact that the relatively healthy hypertrophic vessels in angioid streaks do not appear pathologic. Clinically, moreover, their true appearance is partly hidden by opaqueness of Bruch's layer, and if they are seriously affected, the adjoining choroid

52. Fuchs, A.: Ueber chorioiditis albuminurica, Klin. Monatsbl. f. Augenh. **84**:39, 1930.

53. Gilbert, W.: Die Gefässerkrankungen der Chorioidea, in von Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5.

54. Haab, O.: Atlas und Grundriss der Ophthalmoskopie und ophthalmoskopischen Diagnostik, ed. 2, Munich, J. F. Lehmann, 1897, vol. 7, fig. 75.

has become sclerotic, so that the impression clinically is one of atrophy. It is probable that disease of the isolated artery in angioid streaks is a histologic diagnosis rather than a clinical one.

Disciform Submacular Degeneration.—The Role of a Degenerated Bruch's Layer: As is evident from histologic and clinical data, the degeneration of Bruch's layer begins in the circumpapillary and submacular regions and is complete at the time that the ruptures occur. In the described eyes the development of new tissue was limited to the submacular and the circumpapillary region, though the layer was seriously degenerated even anterior to the equator. In the eye of an old man a circumpapillary and submacular degeneration of the membrane was found without any trace of edema or newly formed tissue. From these facts it may be concluded that the single disease of Bruch's layer does not suffice to explain the appearance of newly formed tissue.

The Role of the Choroidal Vessels: Serious arteriosclerosis may be found at the posterior pole and disease of the choroidal vessels in the peripheral plaque of one of the described eyes, in the sections of the eye of an ape and in an eye affected by herpes, in all of which atrophy of the retina followed, yet newly formed tissue did not develop. A single disease of the vessels does not suffice to explain the appearance of newly formed tissue.

The Nature of Macular Involvement: It is not only in angioid streaks that the macular region degenerates. This area shows a remarkable tendency to degeneration in the retina, pigmented epithelium (various degeneration) and choroid (arteriosclerosis). And in my sections Bruch's layer was also most pathologic in the macular region. Thus deficient nutritional conditions are likely to develop in the macular region comparable with those favoring the development of bone. Mebius,[41] Sacerdotti and Frattin,[55] Liek [56] and Poscharissky [57] ligated the left renal artery and vein, and metaplastic bone developed in the kidney, which still received a blood supply from the capsular vessels.

It is interesting that in my sections also bone was present in the area of macular degeneration. It is possible that a hemorrhage (Verhoeff) or an exudate (Behr and Rintelen) may present the additional stimulus necessary for the formation of the new tissue.

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55. Sacerdotti, C., and Frattin, G.: Sulla produzione eteroplastica dell'osso, Gior. d. r. Accad. di med. di Torino **7**:825, 1901.

56. Liek, E.: Ein weiterer Beitrag zur heteroplastischen Knochenbildung in Nieren, Arch. f. klin. Chir. **85**:118, 1908.

57. Poscharissky, J. F.: Ueber heteroplastische Knochenbildung: Eine pathologisch-histologische und experimentelle Untersuchung, Beitr. z. path. Anat. u. z. allg. Path. **38**:135, 1905.

Varieties of the Conditions of the Choroid: It is interesting that in the families of patients with angioid streaks certain members show a condition of the choroid (Batten,[58] Calhoun,[59] Urrets Zavalía and Obregón Oliva [60]), which other authors (Benedict and Montgomery [61] and Aria [62]) have observed in association with pseudoxanthoma elasticum. In the light of what is known regarding the condition of the choroid, it is interesting that these authors do not speak of arteriosclerosis of the choroid but of a choroidal condition. Batten stated that he has observed choroidal thinning, atrophy and scars. From his observations he concluded that the angioid streaks are definitely of choroidal origin, which is not correct. But his statement that there exists a disease of the choroid, affecting the vessels, which is closely associated with angioid streaks, is still valid and of considerable interest and supports the theory of an independent choroidal disease, different from arteriosclerosis. The diseases seem to be so typical that Batten does not even discuss the possibility of arteriosclerosis. It may be related to the condition described as "peripheral plaque."

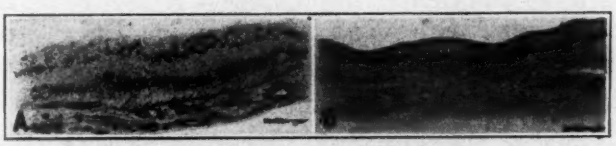

Fig. 45.—*A*, drawing showing the strongly positive iron reaction of Bruch's layer. *B*, drawing showing the positive iron reaction in the basal layers of the corneal epithelium in the limbic region.

The corneal epithelium contained iron at the periphery (fig. 45). Interesting in this respect is the statement of Zondek and Karp [63] that in animals the amount of intracellular iron increases with the age of the cell from infancy up to early middle life. They summarized their view by saying that the iron content of the cell is a measure of its senility. The iron content of the corneal epithelium in the limbic region may account for the vulnerability of this region (marginal corneal ulcers).

58. Batten, R. D.: Angioid Streaks and Their Relation to a Form of Central Choroidal Disease, Brit. J. Ophth. **15**:279, 1931.

59. Calhoun, F. P.: Angioid Streaks of the Fundus Oculi, Am. J. Ophth. **11**:91, 1928.

60. Urrets Zavalía, A., and Obregón Oliva, R.: Estrias angiodeas del fondo del ojo, Arch. de oftal. de Buenos Aires **10**:223, 1935; abstracted, Zentralbl. f. Ophth. **34**:709, 1936.

61. Benedict, W. L., and Montgomery, H.: Pseudoxanthoma Elasticum and Angioid Streaks, Am. J. Ophth. **18**:205, 1935.

62. Aria, S.: Zwei Fälle von Pseudoxanthoma elasticum mit Retinalveränderungen, Zentralbl. f. Ophth. **34**:457, 1935.

63. Zondek, S. G., and Karp, J.: Biochem. J. **28**:587, 1934.

THE NATURE OF THE DISEASE

From the foregoing presentation it is evident that angioid streaks is not a variety of "retinal abiotrophy," as Lindsay Rea [64] recently (1938) stated in his book on neuro-ophthalmology (following Sorsby), since the primary site of the process is in Bruch's layer and the choroid.

It seems possible that the condition of the vessels is secondary to the general hypertension, it being a reaction without any primary pathologic involvement of the vessels, but it must be admitted that this point of view makes it difficult to explain the appearance of the peripheral plaque. The possibility of a specific disease must be left open, particularly since fixation of the material by the solution of calcium may have influenced the microscopic appearance. Thus either the general hypertension or the (functional) inferiority of the elastic tissue must have caused a reactive hyperplasia of the tissue of the arterial wall or a combination of these two factors. The condition of the choroid, though not showing the typical involvement of the elastic tissue, is different from that found in other eyes. It is probable that there exists a similar condition of the arteries and choroid in the cases described by Batten.

The faint staining of the inner elastic layer of the ciliary arteries with hematoxylin may indicate that an incipient process identical with that affecting Bruch's layer is present.

In the center of the pathologic process is Bruch's layer. There is no doubt that there is a degeneration of the elastic fibers of this membrane identical with that seen in pseudoxanthoma elasticum. The theory of a disease of the elastic tissue system is confirmed. But why does this disease limit itself chiefly to Bruch's layer? 1. The reason may be found in the distance of the fibers from their nutrient cells. Wolfrum reported the absence of cells in Bruch's layer, which had already puzzled Fuchs, but described protoplasmic prolongations, extending from the choriocapillary layer toward the membrane. 2. In the skin the elastic fibers which are subject to continuous change in tension are the ones affected. This is also true of the elastic layer of the arteries. Bruch's membrane may be regarded as playing a part in counterbalancing the intraocular pressure, which constantly varies with respiration and the pulse beat. Its degeneration in this disease may be considered a plea in favor of this theory. 3. Little support is found in the eye for an ontogenic theory, since there seems to be little or no difference between the choroid and the layer in the development of their elastic fibers. Wolfrum could confirm Lodato's finding that at the end of the fourth and the beginning of the fifth prenatal month elastic fibers

64. Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938.

develop in a tissue, which is the combined anlage of choroid and sclera. They develop first in the region of the optic nerve and the ciliary body. The membrane can be definitely recognized at a time when only a few elastic elements can be traced in the choroid. I do not believe, however, that from this finding the conclusion can be drawn that the elastic membrane belongs to the first elastic elements which develop in the eye. The membrane may appear as a dark line even in sections that are not stained. Although orcein undeniably is essentially an elastin stain, other tissues take the stain, though they do so in a less degree. Thus the membrane may give a false impression of being colored, just as glass may appear green if the cut surface is presented. 4. Another possibility may be found in a comparative study of the pathologic picture of related diseases.

Diseases in Which Bruch's Layer Is Similarly Affected.—1. The inflammatory and traumatic conditions have been discussed previously.

2. A similar disease of Bruch's membrane, a slight staining with hematoxylin of the elastic layer of some arteries, a degeneration of the elastic fibers of the skin (discussed by Benedict and Montgomery [61]), a positive iron test in the peripheral corneal epithelium and in Bruch's membrane and a disciform degeneration occur both in senility and in angioid streaks.

3. Elective degeneration of Bruch's membrane is further exclusively found in pseudosclerosis (Fleischer), in which, however, the degeneration is due to the presence of a pigment associated with silver (after Vogt [65]). It is interesting that Brouwer and Kuipers [66] described a case of pseudosclerosis and hemochromatosis. In hemochromatosis the pathologic iron pigments are considered a result of an inborn error of metabolism rather than derived from the blood pigment (Sheldon [67]). Pseudosclerosis belongs to a group of diseases which were discussed recently by Brouwer [68] in his paper. The relationship of these conditions has acquired a wider aspect since the study of lipoidosis.

Diseases in Which a Spontaneous Degeneration of a Certain System Occurs.—1. Amaurotic idiocy is an elective degeneration of the ganglion cells of the body. It has been compared and even identified with Niemann-Pick lipoidosis—which is a generalized disease of lipoid metabolism. These lipoidoses are often associated with hemochromatosis.

65. Vogt, A.: Weitere Untersuchungen über die Argyrose des Auges bei Pseudosklerose, Klin. Monatsbl. f. Augenh. **85**:1, 1930.

66. Brouwer, R., and Kuipers, F. C.: Over haemochromatosis met hepatocerebrale degeneratie, Inaug. Dissert., Amsterdam, 1932.

67. Sheldon, J. H.: Haemochromatosis, Lancet **2**:1031, 1934.

68. Brouwer, B.: The Spleen, the Liver and the Brain, Proc. Roy. Soc. Med. **29**:579, 1936.

Such competent authors as Sjövall and Ericsson [69] compared amaurotic idiocy with senile changes.

2. Vitamin A deficiency, which is a disease of metabolism of a special system, is curable; hence other diseases of metabolism may be. The possibility of toxic influences or deficiency in the causation of angioid streaks is supported by Obiditsch's [69a] observation. In a case of sprue with osteoporosis degeneration of the elastic fibers of the skin was found. The fibers were brittle and basophilic and contained lime. Obiditsch did not mention pseudoxanthoma but stated that the only comparable case is that of Jadassohn,[69b] in which there occurred in a boy suffering from osteomyelitis a similar degeneration of these elastic fibers subject to continuous change in tension.

It is a fact that in angioid streaks a specific degeneration of the elastic system occurs at a time when other cells and tissues of the body still are in a good condition. New formation of elastic tissue is still possible, but evidently existing fibers do not reach the age of other tissues. This means a precocious senility of the elastic fibers, which view is supported by the fact that the degeneration occurs especially in regions in which the fibers are subject to continuous change in tension. It is possible that the mechanism of cellular and tissue metabolism which becomes defective in senility may become disturbed in an isolated specific tissue at an earlier age. This conclusion perhaps may be valid also for other disease (amaurotic idiocy). It is not surprising, however, that differences with senility exist, since a presenile disturbance of this metabolism cannot be completely identical with a physiologic deterioration occurring in senility, even if the processes were potentially the same. This degeneration may be due either to a lack of "vitality" of the fibers or to some unknown primary cause, affecting the cellular metabolism. Diabetes (Sugg and Stetson [70]) and osteitis deformans (Terry [71]) occur in the same patient; there may be a relation to iron metabolism. Other diseases of unknown pathogenesis apparently belong to the same group.

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69. Sjövall, E., and Ericsson, E.: The Anatomical Type in the Swedish Cases of Juvenile Amaurotic Idiocy, Acta path. et microbiol. Scandinav., 1933, supp. 16, p. 460.

69a. Obiditsch, R. A.: Ueber patholog-anatomische Veränderungen der Haut bei einem Falle von einheimischer Sprue, Virchows Arch. f. path. Anat. **303**:258, 1938.

69b. Jadasson, I.: Ueber Kaltmetastasen in der Haut, Arch. f. Dermat. u. Syph. **100**:317, 1910.

70. Sugg, E. S., and Stetson, D. D.: Pseudoxanthoma Elasticum Associated with Angioid Streaks of the Retina and Diabetes Mellitus in Sisters, J. A. M. A. **102**:1369 (April 28) 1934.

71. Terry, T. L.: Angioid Streaks and Osteitis Deformans, Tr. Am. Ophth. Soc., 1924, pp. 1-19.

SUMMARY

A case of angiod streaks and pseudoxanthoma elasticum is reported.

The pathologic picture of angioid streaks is described. In the center of the pathologic process there is the degeneration of Bruch's layer, which was demonstrated to be a degeneration of the elastic fibers. Thick-walled arteries different from those in arterio(lo)sclerosis were found. The choroid was not atrophic but thickened (sclerotic).

The degeneration of the elastic fibers of Bruch's layer impairs its translucency, which is the cause of the gray or dotted appearance of the fundus. The completely degenerated membrane acts as a gray veil over the choroid. Angioid streaks are ruptures in the degenerated membrane. The appearance of the fundus varies according to the condition of the pigmented epithelium and the choroid.

In a case of (traumatic) intraocular hemorrhage the other eye should be examined for the presence of angioid streaks. Patients with tapetoretinal degeneration or with an obscure choroidal condition should, together with their families, be subjected to a general physical examination for a possible relation to angioid streaks. Special investigation should be made of the skin. The ophthalmologist is also advised to study the cornea, since a degeneration was found in the limbic region, affecting the basal cells of the corneal epithelium and Bowman's membrane. General examination should not be neglected.

In the future the eyes should be preserved in a neutral solution of formaldehyde or alcohol to prevent solution of calcium and iron.

The nature of the disease is discussed.

REDUCTION OF POSTOPERATIVE COMPLICATIONS IN CATARACT OPERATIONS WITH CORNEOSCLERAL SUTURES

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Ever since Williams,[1] who is generally credited with being the first to advocate the use of conjunctival sutures in cataract extractions, published his paper on this subject in 1869 there have been many advocates of various types of sutures. Among the earliest of these one finds such well known names as H. Knapp,[2] Kalt,[3] Czermak,[4] Vacher[5] and Schweiger[6] and, more recently, Verhoeff,[7] O'Connor[8] and many others. Excellent reviews of the literature on this subject were published by Wolfe and McLeod,[9] Spratt,[10] Ellett[11] and others. Ellett favored Kalt's corneoscleral suture, although he mentioned that "the added wound, though small, introduces an added risk of irritation and infection."

From the Illinois Eye and Ear Infirmary.

Read before the Chicago Ophthalmological Society, Nov. 21, 1938.

1. Williams, H. W.: Cataract Extraction, Arch. Ophth. **1**:102, 1869.

2. Knapp, H.: Reisenotizen, Arch. f. Augenh. **2**:182, 1872.

3. Kalt, E.: Die Kornealnaht nach Extraktion der Katarakt, Arch. f. Augenh. **30**:15, 1895.

4. Czermak, W.: Ueber Extraktion der Cataract ohne Iridectomie mit Naht der Wunde, Wien. klin. Wchnschr. **1**:592 and 618, 1888.

5. Vacher, L.: De l'opération de la cataracte par le procédé à lambeau conjonctival adherent, Ann. d'ocul. **122**:362, 1899.

6. Schweiger, C.: Extraktion mit Lappenschnitt nach unten ohne Iridektomie, Arch. f. Augenh. **36**:1, 1897.

7. Verhoeff, F. H.: Corneo-Sclero-Conjunctival Suture in Operations for Cataract, Tr. Am. Ophth. Soc. **25**:48, 1927.

8. O'Connor, R.: Cataract Extraction by the Undetached Conjunctival Bridge Method After Preliminary Iridectomy, Am. J. Ophth. **17**:809 (Sept.) 1934.

9. Wolfe, O. R., and McLeod, J.: Wound Closure for Intracapsular Extraction of Cataract, Arch. Ophth. **8**:238 (Aug.) 1932.

10. Spratt, C.: Closure of the Cataract Incision, Surg., Gynec. & Obst. **60**:573 (Feb.) 1935.

11. Ellett, E. C.: Use of the Suture in Extraction of Cataracts, Arch. Ophth. **17**:523 (March) 1937.

Recently the popularity of suturing cataract wounds has increased rapidly, but there are still a good many operators who are content to leave conjunctival flaps unsutured. A surgeon is usually desirous of having some proof of the efficacy of a change in surgical technic before altering a well established method. One finds in the literature many statements as to the merits of the various types of sutures, but insufficient proof is offered to warrant a universal change in technic. Statements like that of Spratt [10] are rather common in the literature:

> It is a well recognized principle that unless drainage is indicated operative wounds should be sutured and not left for nature unaided to make closure. Rapid healing is thus obtained, infection is avoided and the danger of postoperative hernia is reduced to a minimum. Incisions into the cranial cavity, abdomen, or thorax are sutured. Why should an incision in the eye remain unsutured?
>
> After the ordinary cataract section, the edges of the wound tend to adhere. We all know, however, from past experience that such closure is not free from complications, such as delayed union, reopening of the wound by coughing and straining, downward growth of epithelium into the anterior chamber (Elschnig has demonstrated this), prolapse of iris, vitreous loss, expulsive hemorrhage and late infection.

Spratt, like many others, asks one to accept the bare statement that postoperative complications are reduced when sutures are used.

Feeling that these statements were correct because groups of our patients on whom corneoscleral sutures had been used did show a definite reduction in the relatively common postoperative complication of prolapse of the iris, with its tendency to produce a slowly healing, irritable eye and frequently secondary glaucoma, we decided to make an impartial examination of the hospital records of the Illinois Eye and Ear Infirmary to see if we could find proof of this assumption. We made a study of the relative efficiency in preventing postoperative complications in cases that fell under three groups: group 1, those in which conjunctival flaps were used without sutures; group 2, those in which conjunctival flaps were used with sutures, and group 3, those in which corneoscleral sutures were used. One hundred and fifty histories for each group were tabulated. As the corneoscleral sutures were used periodically, the histories for the other groups were taken in the order in which they appeared in the files, covering the same periods during which corneoscleral sutures were used. As these cases were gathered over a period of two years and a large percentage of the operations was done by residents and interns who rotate between the various services, spending an equal amount of time in each, a large part of the surgical work in each group was done by the same men. The rest was done by members of the attending staffs, who of course do not rotate but are considered to have equal ability in surgical technic.

In the group in which corneoscleral sutures were used, no cases were excluded, because in those in which postoperative complications developed the patients did not have an associated pathologic condition, such as diabetes, vascular hypertension, damage from previous iritis and glaucoma. However, in groups 1 and 2, in order to remove any question that such a pathologic condition might have contributed to postoperative complications, all cases in which postoperative complications developed in the presence of an associated pathologic process were excluded.

As the records were written up by various interns and residents under somewhat variable conditions, some are not as full and complete as they would have been had they been prepared by one group with the intention of publishing the results. Again, the percentages of postoperative complications are probably a little higher in our series of cases than would be expected in a private hospital where day and night private nursing care is the usual procedure for the first few days.

The postoperative complications considered in this paper are: (1) prolapse of the iris or vitreous, or both; (2) delayed closure of the anterior chamber, and (3) hyphema. The misfortunes following prolapse of iris and vitreous are well known to all. The consequences of delayed closure of the anterior chamber have been pointed out by several authors, recently by Perera [12] who has shown that hypotony favors the growth of epithelium into the eye. As to hyphema, not much is found in the recent literature, but Vail [13] in 1933 termed it "an always annoying and often serious complication" and further stated:

> Complications of hyphema and wound reopening are: iritis, adherent iris, blood particles remaining in the vitreous, organized secondary membrane, detached choroid, glaucoma, and blood staining of the anterior hyaloid membrane or cornea.

However, no serious after-effects of hyphema were noted in any of the records we examined for this paper.

Our investigation showed that these postoperative complications were greatly diminished in the group in which conjunctival sutures were used and further reduced in the group in which corneoscleral sutures were employed. This is shown in tables 1, 2 and 3.

TYPE OF CORNEOSCLERAL SUTURE USED

Most of the corneoscleral sutures used in our group consisted of two transverse bites, one in the sclera and the other in the cornea, equidistant from the limbus with about 2.5 to 3 mm. separation. The length of bite was also about 2.5 to 3 mm. A double-armed suture

12. Perera, C. A.: Epithelium in Anterior Chamber After Operation and Injury, Am. J. Ophth. **21**:615 (June) 1938.

13. Vail, D. T., Jr.: On Hyphema After Cataract Extraction, Tr. Am. Ophth. Soc. **31**:496, 1933.

TABLE 1.—*Data on Cases in Group 1 in Which Postoperative Complications Occurred* *

Patient	Iridectomy	Extracapsular or Intracapsular Extraction	Loss of Vitreous During Operation	Postoperative Complications	
				At First Dressing (48 to 72 Hr.)	Later
F. J.	Complete	Extracapsular	None	Prolapse of iris	2d prolapse on 6th day
O. S.	Complete	Extracapsular	None	Chamber not formed	Chamber not formed until 8th day; hyphema on 13th day; glaucoma 48 days later
M. S.	Complete	Extracapsular	None	Chamber not formed	
M. T.	Complete	Extracapsular	None	Wound open, vitreous extruding; prolapse of iris	
M. M.	None	Intracapsular	None	Chamber not formed	Chamber not formed on 7th day
A. S.	None	Extracapsular	None	Hyphema	
E. M.	Complete	Extracapsular	None	Incarceration of iris; chamber not formed	
V. V.	Complete	Intracapsular	Loss of vitreous	Wound gaping; vitreous protruding	
H. C.	Complete	Intracapsular	Not recorded	Chamber not formed	
M. C.	Complete	Extracapsular	Loss of vitreous	Prolapse of vitreous	
J. N.	Complete	Extracapsular	None	.....	Hyphema on 5th day
C. B.	Complete	Intracapsular	None	Small hemorrhage	
M. A.	Complete	Intracapsular	None	Chamber not formed	Chamber not formed in 1 week
J. M.	Peripheral	Extracapsular	None	Small hyphema	
S. M.	Complete	Extracapsular	None	Wound open; prolapse of vitreous and iris	
J. B.	Complete	Extracapsular	None	Chamber not formed	
M. P.	None	Extracapsular	None	.....	Iris incarcerated on 5th day
M. H.	Complete	Extracapsular	None	Slight prolapse of iris	
N. T.	Complete	Extracapsular	None	Slight prolapse of iris; slight hyphema	
A. B.	Complete	Intracapsular	None	Chamber not formed	
J. E.	Complete	Extracapsular	None	Hyphema	
A. L.	Complete	Extracapsular	None	Chamber not formed	Prolapse of iris; wound gaping on 5th day
P. M.	Complete	Extracapsular	None	Prolapse of vitreous	
E. R.	Complete	Extracapsular	None	Chamber not formed	
A. P.	Complete	Extracapsular	None	Hyphema	
E. B.	Complete	Extracapsular	None	Hyphema	
J. W.	Complete	Extracapsular	None	Hyphema	
B. L.	Complete	Extracapsular	None	Hyphema	
M. H.	Not recorded	Extracapsular	None	Chamber not formed	Chamber not formed on 5th day
A. O.	Complete	Extracapsular	None	Prolapse of iris	

SUMMARY OF COMPLICATIONS

	Cases
Prolapse of iris or vitreous, or both; or incarceration of iris in wound	12 (8%)
Observed at first dressing	10
Wound open with prolapse of vitreous and iris	2
Prolapse of vitreous	3
Prolapse of iris	4
Incarceration of iris	1
Observed on 5th day	2
Prolapse of iris	1
Incarceration of iris	1
Hyphema	9 (6%)
Observed at first dressing	7
Observed later	2
On 5th day	1
On 14th day	1
No anterior chamber present at first dressing	11 (7.4%)
Not formed until 5th day	1
Not formed until 7th day	3

* The histories of 150 cases in which no sutures were used to secure the conjunctival flaps were examined. All of these cases were from one service, and the surgical work was done by various operators, consisting of members of the attending and resident staffs. Nine cases in which complications occurred postoperatively were excluded because of an associated pathologic process. Of the 150 cases recorded, complete iridectomy was done in 136 and peripheral iridectomy in 12, while iridectomy was not performed in 2. Extracapsular extraction was done in 129 cases and intracapsular extraction in 21. Loss of vitreous occurred at operation in 13 cases.

TABLE 2.—*Data on Cases in Group 2 in Which Postoperative Complications Occurred* *

Patient	Iridectomy	Extracapsular or Intracapsular Extraction	Loss of Vitreous During Operation	Postoperative Complications	
				At First Dressing (48 to 72 Hr.)	Later
L. M.	Complete	Intracapsular	Loss of vitreous	Prolapse of iris and vitreous	
O. B.	Complete	Extracapsular	None	Slight hyphema	Prolapse of iris (date not recorded)
E. S.	Complete	Extracapsular	None	Chamber not formed	
O. S.	Complete	Extracapsular	None	Prolapse of iris	
H. H.	Complete	Extracapsular	None	Slight hyphema	
E. K.	Complete	Extracapsular	None	Hyphema	
G. P.	Peripheral	Extracapsular	Loss of vitreous	.....	Prolapse of vitreous on 12th day
B. W.	Complete	Intracapsular	None	Chamber not formed	
L. Y.	Peripheral	Intracapsular	Loss of vitreous	Slight hyphema	
A. B.	Periphreal	Intracapsular	None	.....	Partial prolapse of iris on 6th day
J. Y.	Peripheral	Intracapsular	None	Chamber not formed	
G. P.	Complete	Extracapsular	None	.....	Hyphema on 10th day
H. M.	Complete	Extracapsular	None	Wound open; prolapse of vitreous and iris	Glaucoma
W. R.	Peripheral	Extracapsular	None	Chamber not formed	
J. K.	Peripheral	Intracapsular	None	Hyphema	
W. B.	Complete	Extracapsular	None	.....	Hyphema on 5th day
M. E.	Complete	Extracapsular	None	Chamber not formed	Small hyphema on 4th day
W. S.	Complete	Extracapsular	None	Marked hyphema	
M. E.	Complete	Extracapsular	None	Chamber not formed	
M. O.	Complete	Extracapsular	None	Anterior chamber filled with blood	
T. B.	Complete	Extracapsular	None	Chamber not formed	Prolapse of iris on 11th day
W. V.	Complete	Extracapsular	None	Hyphema	
B. P.	Complete	Extracapsular	None	Hyphema	
M. F.	Not recorded	Intracapsular	Loss of vitreous	.....	Hyphema on 7th day
J. W.	Complete	Extracapsular	Loss of vitreous	.....	Slight prolapse of vitreous at 1 month
L. S.	Complete	Extracapsular	None	Chamber not formed	Prolapse of iris on 14th day
G. S.	Complete	Intracapsular	None	.....	Hyphema on 7th day
J. J.	Complete	Extracapsular	None	Hyphema	
M. J.	Complete	Extracapsular	None	Slight hyphema	
M. B.	Complete	Extracapsular	None	Chamber not formed; hyphema	
J. C.	Complete	Extracapsular	None	Prolapse of iris	

SUMMARY OF COMPLICATIONS

	Cases
Prolapse of iris or vitreous, or both	10 (6.6%)
Observed at first dressing	4
Observed later	6
Prolapse of iris	4
Observed on 6th day	1
Observed on 10th day	1
Observed on 13th day	1
Date not recorded	1
Prolapse of vitreous	2
Observed on 11th day	1
Observed 1 mo. later	1
Hyphema	17 (11.3%)
Observed at first dressing	12
Observed later	5
Observed on 4th day	3
Observed on 7th day	1
Observed on 10th day	1
No anterior chamber present at first dressing	9 (6%)

* The histories of 156 cases in which the conjunctival flaps were secured with conjunctival sutures were examined. These cases were taken from all services, and the surgical work was handled by various operators, consisting of members of the attending and resident staffs. Six cases in which complications occurred postoperatively were excluded because of an associated pathologic process. Of the 150 cases recorded, two sutures were used in 67 and one suture in 83. Complete iridectomy was performed in 111 cases and peripheral iridectomy in 39. Extracapsular extraction was done in 99 cases and intracapsular extraction in 51. Loss of vitreous occurred at operation in 22 cases.

TABLE 3.—*Data on Cases in Group 3 in Which Postoperative Complications Occurred* *

Patient	Iridectomy	Extracapsular or Intracapsular Extraction	Loss of Vitreous During Operation	Postoperative Complications	
				At First Dressing (48 to 72 Hr.)	Later
M. P.	Complete	Intracapsular	Loss of vitreous	Slight hyphema	
F. L.	Complete	Extracapsular	None	.....	Slight prolapse of iris on 7th day
E. E.	Complete	Extracapsular	None	.....	Prolapse of iris on 14th day
M. E.	Complete	Extracapsular	None	.....	Hyphema on 8th day
H. G.	Complete	Extracapsular	None	.....	Hyphema on 6th day
M. B.	Complete	Extracapsular	None	.....	Hyphema on 6th day
B. K.	Not recorded	Extracapsular	None	Incarceration of iris	
E. W.	Peripheral	Extracapsular	None	Prolapse of iris	
K. M.	Complete	Intracapsular	None	.....	Slight hyphema on 3d day
L. M.	Peripheral	Intracapsular	None	Prolapse of iris	
V. H.	Peripheral	Intracapsular	None	Hyphema	
B. K.	Complete	Extracapsular	None	Hyphema	
L. P.	Complete	Intracapsular	None	Chamber not formed	
H. K.	Peripheral	Intracapsular	None	Chamber not formed	
P. G.	Peripheral	Extracapsular	None	Prolapse of iris	
T. B.	Complete	Extracapsular	None	Chamber not formed	
R. L.	Complete	Intracapsular	None	Chamber not formed	

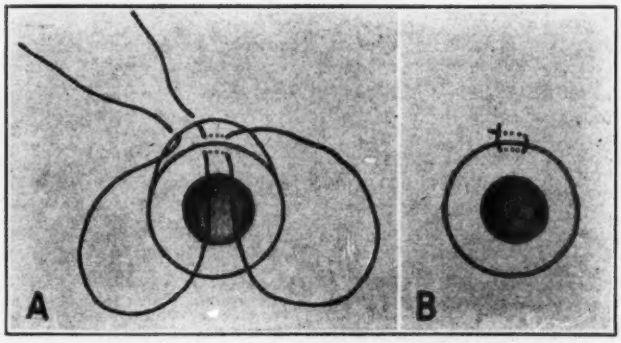
SUMMARY OF COMPLICATIONS

	Cases
Prolapse of iris or incarceration of iris in wound (no instances of prolapse of vitreous)	6 (4%)
Observed at first dressing	4
Observed later	2
Observed on 7th day	1
Observed on 14th day	1
Hyphema	7 (4.6%)
Observed at first dressing	3
Observed later	4
Observed on 6th day	2
Observed on 3d day	1
Observed on 8th day	1
No anterior chamber present at first dressing	4 (2.6%)

* The histories of 150 consecutive cases in which corneoscleral sutures were used were examined. The majority were from the Thomas D. Allen service, and the surgical work was done by various operators, consisting of members of the attending and resident staffs. No cases were excluded from this group. Of the 150 cases, Verhoeff sutures with vertical needle tracts placed prior to incision were used in 30 (earlier cases) and sutures such as are described were used in 120. Complete iridectomy was done in 105 cases and peripheral iridectomy in 30; in 3 cases iridectomy was not performed, and there was no record concerning 12. Extracapsular extraction was done in 64 cases and intracapsular extraction in 85. The type of operation was not recorded for 1 case. Loss of vitreous occurred at operation in 12 cases.

was found most suitable, because both needles could be introduced in the same direction, allowing only one fixation to be made on a lateral rectus muscle the plane of action of which was opposite to the direction of force exerted by the needles. The suture, of course, is placed prior to making the corneal incision. This type of suture has been recommended by several authors, and recently a good article has been published by Stallard [14] of London. He reported 107 cases in which this suture was used without a single postoperative mishap.

As an added precaution against the accident of opening the anterior chamber while removing the suture (this happened in 3 of our earlier cases), we have lately been in the habit of dissecting a small Van Lint flap about 10 mm. along the limbus and inserting both ends of the suture through this, so as to tie the knot over the flap, which forms a pad between the knotted part of the suture and the wound, as shown in the accompanying illustration. This allows the suture to be grasped



A, suture in position, ready for corneal section. (The flap in this drawing is somewhat larger than is generally made.) *B*, appearance of suture at completion of operation.

easily, and any traumatism in severing it would be limited to the conjunctival pad beneath.

We like to use 12 mm. curved round needles with small, unsplit eyes and black braided silk, size 0.006. The needle holder should have a small tip that will hold the delicate needle firmly without breaking it.

The apparent opinion of some operators is that there is considerable difficulty in the use of corneoscleral sutures. The following quotation is from a recent article by Gailey:[15]

> As to sutures, any suture is better than none. Conjunctival sutures, even six or seven, are inadequate. It would seem that closure of the deep wound is the

14. Stallard, H. B.: A Corneo-Scleral Suture in Cataract Extraction: Its Technique and Advantages, Brit. J. Ophth. **22**:269 (May) 1938.

15. Gailey, W. W.: Some Problems Encountered in Cataract Surgery, Am. J. Ophth. **21**:855 (Aug.) 1938.

proper solution. Various types of corneo-scleral sutures have been tried, but none has been found that is easy of application and at all times pleasing. The difficulty may be in not adhering to one technique long enough to grow really proficient in it.

Although some patience and perseverance are required for the first few cases, a little practice, as a rule, will enable any one with a reasonable amount of skill to use the type of suture described here. After experimenting somewhat with various kinds of sutures, we felt definitely that this suture gave us all the advantages of any other and was the simplest one to use.

SUMMARY AND CONCLUSION

A comparison of the complications in the three groups, as presented in table 4, is unquestionably favorable to the corneoscleral suture, especially in view of the fact that 15 cases were excluded from groups 1 and 2:

TABLE 4.—*Comparison of Complications in the Three Groups*

	Prolapse of Iris and Vitreous; Incarceration of Iris	Hyphema	Anterior Chambers Not Reformed
Group 1	12 cases (8.0%)	9 cases (6.0%)	11 cases (7.3%)
Group 2	10 cases (6.6%)	17 cases (11.3%)	9 cases (6.0%)
Group 3	6 cases (4.0%)	7 cases (4.0%)	4 cases (2.6%)

The factors possibly contributing to complications in these 15 cases are presented in the following outline:

Group 1 (9 cases excluded)

- Prolapse of iris 9
  - High blood pressure 2
  - Diabetes 2
  - Prolapse probably due to trauma on way to dressing room 1
  - Expulsive hemorrhages 2
  - Hyphema with hypertension 2

Group 2 (6 cases excluded)

- Prolapse of iris 4
  - Prolapse while dressing 1
  - High myopia 1
  - Diabetes 1
  - History of previous iritis 1
- Hyphema with hypertension 1
- Expulsive hemorrhage 1

No cases were excluded from group 3. Also, there were postoperative factors in the following 3 cases in group 3 which might have

resulted in complications had the wounds not been firmly closed by corneoscleral sutures:

1. In K. M., aged 67, with a systolic blood pressure of 210 and a diastolic pressure of 110, bronchitis and a severe cough developed on the fourth day.

2. M. K., aged 75, on the third day tore off his bandage, and the retaining lid suture came with it. He was out of bed walking around when this was discovered.

3. C. F., aged 70, on the first day, a few hours after operation, tore his bandage off and walked around the room.

It might also be of interest to point out that many more intracapsular operations were done in the group in which corneoscleral sutures were used than in the other two groups (21 in the group in which sutures were not used; 51 in the group in which conjunctival sutures were used, and 80 in the group in which corneoscleral sutures were used). The question arises if there would not have been more postoperative complications in groups 1 and 2 if the same number of intracapsular operations had been done in each.

Our statistics point to the following advantages of the corneoscleral suture:

1. The number of postoperative prolapses are reduced, and when prolapse does occur it is smaller.

2. The occurrence of hyphema is reduced.

3. Anterior chambers are formed earlier.

4. Prolapse of the vitreous is eliminated.

5. If vitreous is lost during operation, the wound is closed securely after delivery of the lens and no further loss is feared.

6. When the ends of the suture are drawn taut, firm apposition of the surfaces of the wound is obtained even before tying, allowing irrigation when indicated and toilet of the wound to be completed in safety. If added safety is desired, these manipulations can be accomplished after tying the suture.

7. In cases in which patients cough, vomit or become violent, the eyes are much more safe.

8. Patients are permitted earlier and greater freedom of movement. This is important in aged patients. (Several were allowed to sit up in bed twenty-four to forty-eight hours after operation.)

9. It is especially valuable for the younger ophthalmic surgeon in his early surgical practice, because his incisions are likely to be less even, and the delicate touch which comes only after much practice may be somewhat lacking.

10. It is valuable for patients who cannot receive the benefit of private nursing and are consequently more likely to be restless, thus inviting the danger of traumatism.

11. It is particularly well adapted to the intracapsular method, as there is more tendency to prolapse of vitreous when the posterior lens capsule is torn free from the suspensory ligament.

Only about half of the patients in each group of cases returned for final refraction, and this small number was considered of little value in determining the undecided question as to whether or not the averages for postoperative astigmatism are lower in the group in which corneoscleral sutures were used than in those cases in which this type of suture was not used. However, our averages in diopters of astigmatism for those patients who did return were: group 1, 2.31 diopters; group 2, 1.94 diopters, and group 3, 1.82 diopters.

No bad effects of any sort resulted from the use of corneoscleral sutures. The only disadvantages in their use, if they must be called such, are the necessity of a little practice before they can be deftly and quickly inserted; and for the experienced surgeon, about two minutes longer time spent on the operation.

TREATMENT OF DISEASES OF THE EYE WITH GRENZ RAYS

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Many forms of radiant energy are being used in this country and abroad for the treatment of diseases of the eye, with varying and sometimes controversial results. Among them are grenz rays, which have been used for the past ten years in Europe with reported success. Already an extensive literature has appeared on the subject. This form of therapy was adopted by me after first hand observation of its technic and results, and a report of my experience follows, together with a brief discussion of the physics and the biologic and clinical effects of grenz rays.

PHYSICS AND BIOLOGIC EFFECT

Grenz rays are effective, though not very penetrating, rays which are absorbed in the superficial tissues. They are sometimes called Bucky rays, after Bucky, who established their value as a therapeutic agent. They are electromagnetic oscillations of about 2 angstrom units which produce characteristic clinical and biologic manifestations. Their position in the electromagnetic spectrum is between the ultraviolet rays and the roentgen rays. Grenz rays are generated in a hot cathode vacuum tube, as are roentgen rays, but at a much lower voltage. These rays have long wavelengths, and as such are soft rays, which do not penetrate the wall of the ordinary roentgen ray tube because of the thickness of the glass. They are therefore generated in a tube provided with an especially thin window, which is energized by an especially constructed apparatus.

The quality of grenz rays is measured in half value layer of aluminum, as is that of roentgen rays, and the quantity is measured in roentgens. These rays are absorbed in air, and the average absorption of soft rays in tissue is about equal to that in water. Bucky, by applying the theoretic determination of Frank, calculated the ratio of the half value layer in water and in different parts of the skin to the half value layer in aluminum and found for certain conditions at a voltage of 10 kilovolts and 0.03 mm. of aluminum, half value layer (wavelength

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of 1.7 angstrom units), that half the radiation is absorbed in 0.47 mm. of water, in 0.52 mm. of epidermis and in 0.77 mm. of subcutaneous tissue. Approximately 60 per cent of a grenz ray of 10 kilovolts is able to penetrate 0.5 mm. of skin tissue; 30 per cent is able to penetrate 1 mm., and 14 per cent, 2 mm. This superficial absorption seems to fit the rays admirably for the treatment of superficial lesions of the eye, for the danger of producing untoward effects is virtually negligible, except in inordinate doses.

The biologic effect of grenz rays is similar in some respects to that of roentgen rays. Slight erythema of the skin can be produced with approximately 300 r. The degree of erythema depends on the quality or hardness of the rays and on the number of roentgens delivered to the surface of the skin. If a sufficient dose is given, desquamation may follow the erythema, and pigmentation may be the end result. The intensity of pigmentation depends on the amount of pigment in the skin before treatment and shows a constant relation to the erythema; the pigmentation may remain for years. With rather high doses erythema may appear within a few hours, and with still higher or experimental doses a bluish red edema with slight burning pain may be produced. I have never observed any comparable response of the conjunctiva or cornea to massive doses, and it is my belief that mucous membranes are much more tolerant.

Hausser and Schlechter investigated the erythema produced by grenz rays and roentgen rays and plotted a flat curve for that produced by grenz rays and a steep curve, which was identical, for the erythema produced by roentgen rays and ultraviolet rays of 313 angstroms. Wilhelmy showed that the erythema curve for medium hard roentgen rays is nearly the same as that for grenz rays. This work was recently checked by Leitner, of London.

Bucky, and subsequently many investigators, observed that from ten to thirty minutes after grenz irradiation the number of leukocytes in the peripheral blood was reduced by about one third and that after an equal period of time the original number was restored. Roentgen irradiation may produce a decrease of leukocytes also, but this occurs several hours after irradiation. Bucky showed further that the decrease of leukocytes did not occur if the part irradiated was previously sympathectomized. This observation is the basis for Bucky's explanation of the effect of grenz rays on the vegetative nervous system and is the indication for their employment in the treatment of constitutional diseases.

Untoward reactions with high doses of grenz rays may be experienced, and the range of safety has not been accurately determined. But it has been adequately shown that when compared with roentgen rays the use of these rays of long wavelength is relatively free from danger.

Several times the erythema dose may be administered at one sitting without causing epilation, but if the dose is sufficiently large and penetrating alopecia may result. Dermatologists in Germany have used as much as from 4,000 to 8,000 for a single dose without producing incurable ulcers. Sequelae are uncommon. A number of cases have been recorded in which massive doses produced telangiectasia and atrophy. It has been claimed that there are no cumulative effects as are experienced with roentgen rays, but this is not generally accepted, and in my experience mildly untoward reactions seem to have resulted from closely repeated relatively small doses.

Because of the delicacy of the eyeball and of the skin of the eyelid, I was careful to select doses which could be well tolerated. In the beginning small doses were given, but later the usual practice was to give 452 r with 0.024 mm. of aluminum, half value layer, twice weekly until five treatments had been administered. In urgent cases treatments were given daily or every other day. The apparatus was checked at intervals for quantity and quality of output of rays. Because of the pigmentation, which always appeared in brunette patients, the use of the rays for the treatment of various conditions of the eyelid was abandoned. On two occasions it was thought that an overdose had been given: The patients had received four series of five treatments each of 452 r with intervals of rest of two weeks. In these cases there seemed to be increased inflammation of the conjunctiva and episcleral tissue, manifest by increased engorgement of the vessels and by pain. Destruction of the corneal tissue itself with subsequent scarring and opacification of the crystalline lens have never been observed. The outstanding favorable effect was analgesia, which was well demonstrated in most cases. In many cases in which a definite improvement was not observed the analgesia brought about was gratifying to the patient. Histologic examination of corneal tissue of rabbits after four series of treatments had been given failed to show any pathologic change.

RESULTS OF EXPERIMENTS

In order to gain an understanding of the effectiveness of grenz rays and to obtain a guide for the regulation of dosage, I conducted a number of experiments on the eyes of rabbits.

That local antibody formation of the cornea can be stimulated by grenz rays was demonstrated by Thompson, Gallardo and myself in a series of experiments published last year.

In a series of unpublished experiments Gallardo and I have shown that grenz rays have a definite effect on the course of ulcers induced in corneas of rabbits. In two series of rabbits, the first consisting of 4 rabbits and the second of 6, ulcers were induced in the corneas of

both eyes with the injection of a strain of staphylococcus of known pathogenicity; the right eyes were then irradiated, and the ulcers of the left eyes were allowed to run their courses untreated. The ulcers of the treated eyes were decidedly smaller and less penetrating and were accompanied by much less reaction. The healing was much more rapid in these eyes than in the untreated ones, and the scar formation was less opaque.

Krasso, of Vienna, conducted a number of experiments in which she also studied the effect of grenz rays on the eyes of rabbits. When a dose of from 980 to 4,000 r of 0.02 mm. of aluminum, half value layer, was given, edema of the epithelium resulted. Later, treatment of the same eye with 4,000 r of 0.025 mm. of aluminum, half value layer, resulted in a disk-shaped opacity of the cornea. A third treatment of 4,000 r of 0.031 mm. of aluminum, half value layer, was given, and necrosis of the superficial layers of the cornea followed, which terminated in scar formation of the cornea.

CLINICAL RESULTS

My clinical experience with grenz rays extends over the past five years, during which time 302 patients were treated, and more than 1,200 treatments were given.

After the first few months I accepted for treatment only persons with surface lesions of the eyeball, cornea, bulbar conjunctiva, episclera and sclera. I did not attempt to treat diseases of the palpebral conjunctiva because of the difficulty of exposing this membrane to the rays. Save for a few patients with blepharitis and dermatitis venenata, those with diseases of the eyelids were not accepted, because of discoloration which frequently resulted from the production of pigment. All patients were examined before and carefully observed during the treatments, and other measures, such as the use of atropine and dressings when indicated, were continued during the course of the irradiation.

The most striking response was observed in ulcers and infiltrates of the cornea, and in these instances there was convincing proof of the effectiveness of grenz rays. Thirty-two patients with various types of ulcers of the cornea were treated, and there was stimulation of healing apparent in practically all catarrhal and small marginal ulcers in from one to four days. In cases of severe ulcers protective dressings were applied, and some of the healing must be credited to this measure. Four patients with exceedingly severe nonspecific corneal ulcers in which the purulent disintegration involved much of the stroma were treated with grenz rays after all other measures had proved futile, and prompt relief from pain and healing were observed. Trachomatous ulcers seemed to respond promptly. Eleven patients with dendritic ulcers were treated.

and the results were inconclusive. Curettage and cauterization were done on most of these patients before the effect of grenz rays could be observed. Three patients with recurrences after curettage were treated, and the process was controlled. In this connection it should be pointed out that irradiation is a slower method of treatment than surgical intervention and that time is required for results to appear. Two patients with abscess of the cornea were treated, and both showed continued localization and gradual healing, with a minimum of opacification resulting. Rodent ulcers (Mooren's ulcer) were not treated. Krasso, however, reported favorable results in 3 patients with this type of ulcer. One patient with a late hypopyon ulcer due to the pneumococcus received three treatments on alternate days without apparent benefit.

The next most impressive results were obtained in cases of episcleritis and scleritis. Sixty patients with episcleritis were treated, and results which could be ascribed to the therapy were observed in 48, or 80 per cent. Six patients were not benefited in the least. It was most difficult to evaluate the therapy in these cases, however, because of the usually extremely variable duration of the disease and of the apparent variable and indefinite causation. In most cases there appeared to be a contraction of the vessels and a fading of the lesion within a week after the treatments were begun. Four patients with scleritis were treated, and all showed definite response.

A great variety of types of nonulcerative keratitis were treated, and constancy of results was not observed. Twenty-nine patients with superficial punctate keratitis of the type characterized by minute staining lesions of the epithelium were given sufficient treatments to produce results. Most of the patients obtained relief from the discomfort experienced; the condition of a few improved, and that of 6 cleared during the course of treatment. Five patients with superficial punctate keratitis (Fuchs) were benefited, but complete cure was not obtained. Six patients with phlyctenular keratitis were treated, and healing of the lesions which might be ascribed to the grenz rays was not observed. Six patients with epithelial dystrophy of the cornea were treated, and the vesicular lesions of the epithelium of 2 were definitely removed and after two years have not recurred. The accompanying endothelial dystrophy in these 2 patients who received treatment over periods of six and nine months was unchanged. Two of the patients were perhaps benefited, and 2 did not have full series of treatments. Four patients with herpetic keratitis were helped through relief from symptoms. Three patients with disciform keratitis, 2 with sclerosing keratitis and 3 with band keratitis were not benefited.

Thirteen patients with vascularized keratitis, a grouping which includes various types of parenchymatous involvement, were not uni-

formly benefited. Their distressing symptoms, however, were relieved in part, and in some instances the vessels were reduced in size. This effect on blood vessels was noted in many of the cases in which there were various kinds of vascularization. The pannus of trachoma was reduced, and the newly formed vessels in cases of corneal transplants were reduced in caliber. The nebulosity of the corneal transplants, however was not appreciably affected. Old scars of the cornea were not altered.

In the several cases of dermatitis venenata of the eyelids improvement was not observed, but it should be said, however, that treatments were not continued long enough to produce results. Fissures of the skin

Therapeutic Results with Grenz Rays

Conditions Treated	Number of Cases	Number of Treatments	Results
Ulcer of cornea	82	133	Healing effect or cure
Abscess of cornea	2	10	Healing effect or cure
Dendritic keratitis	11	48	Not uniform
Trachomatous keratitis	8	32	Favorable in ulcers and pannus
Phlyctenular keratitis	6	27	Relief of symptoms
Vascularized keratitis	13	63	Not uniform
Superficial punctate keratitis (Fuchs)	5	57	Not uniform
Superficial punctate keratitis	29	102	Not uniform
Band keratitis	3	19	Not helpful
Disciform keratitis	3	12	Not helpful
Sclerosing keratitis	2	17	Not helpful
Herpetic keratitis	2	3	Relief from symptoms
Keratitis profunda	4	20	Not helpful
Vernal conjunctivitis	7	41	Healing of limbal hyperplasia
Epithelial dystrophy of cornea	6	47	Not uniform
Vascularized and nebulous corneal transplants	13	61	Not uniform
Keratitis (unclassified)	28	125	
Conjunctivitis (various types)	24	67	Not uniform
Episcleritis	60	237	Healing effect or cure
Scleritis	4	53	Healing effect or cure
Blepharitis	9	23	Not uniform
Dermatitis venenata of eyelids	5	17	Not helpful
Miscellaneous	27	61	
Total	302	1,253	

at the canthi were treated, and healing was aided. The stimulation of pigment formation by grenz rays was made use of in the treatment of a Negro child who had areas of diminished pigment of the skin of the orbital regions. A number of patients who complained of painful eyes associated with glaucoma, hyperthyroidism and sun bathing were treated for the analgesic effect without benefit. One patient with keloids of the lids was treated, and no effect was observed. Three patients with postoperative pterygiums were treated, and scarring was held to a minimum and recurrence was apparently controlled.

COMMENT

The evaluation of clinical results of any form of treatment calls for close observation, shrewd judgment and a mature experience in ophthalmology. To attribute a cure or a shorter course of disease to a new form

of therapy may be denying the defense or healing mechanism of tissue. The worker with a new form of treatment, through his intimacy, is prone to be prejudiced and to be more impressed by the happy results than by the failures. Because of the many factors and variables concerned, a report of this type should be founded only on a large series of patients and on experience gained with the treatment of many patients with similar disease entities. I endeavored to check results on patients with disease of both eyes whenever possible by treating one eye and using the other as a control, and, in addition, my notes and impressions were compared with those of the referring physicians.

This report does not offer final evidence of the value of grenz rays in ophthalmology. The experience gained is simply regarded as indicative, and my impression is that the treatment is helpful in inflammatory diseases and in many conditions for relieving distressing symptoms. There was little evidence to indicate any healing effect in the dystrophic diseases of the cornea. That it is effective in treating radiosensitive new growths is adequately supported by the literature. I observed two small basal cell epitheliomas respond to the treatment which was given in another laboratory.

The question is frequently asked: How much more effective are grenz rays than the methods of treatment commonly employed? In evaluating results, two thoughts were kept constantly in mind: 1. Can the course of the disease be attributed to grenz rays, and if so, are the rays aborting the disease, shortening the course or affecting it at all, or are they retarding progress? 2. Are these rays succeeding better than the usual forms of therapy? The results tabulated were arrived at after consideration of these questions. The value of grenz rays in the treatment of ulcers was outstanding, and in chronic inflammatory conditions the therapy was most successful, succeeding after all other methods had failed. There is no efficacious treatment for episcleritis and scleritis, unless it is sulfanilimide, so that grenz rays scarcely have a competitor, save perhaps radium and roentgen rays. The scarring produced by escharotic agents is avoided, the patient is more comfortable, and the treatments are easily administered.

It is my opinion that grenz rays succeed in the treatment of surface lesions of the eye as well as other forms of irradiation and have the important factor of safety to commend them. The possible damaging effects of roentgen rays and of radium are well known, and one does not feel justified in running any risk with these modalities in the treatment of nonmalignant disease of the eye, for their limits of safety have not been demonstrated. Ultraviolet rays are entirely superficial in their effect, are of disputed efficacy and are painful. It seems probable that grenz rays will replace other forms of radiant energy in their field of usefulness.

With continued work and experience, the sphere of usefulness of grenz rays, which seems promising indeed, will be better defined. Many variable factors remain for study, and much experimentation will have to be carried on before their maximum efficacy will be known.

SUMMARY

The rationale for the use of grenz rays in the treatment of disease of the eye is indicated.

The physics and the biologic and clinical effects of grenz rays are discussed briefly.

Results of experiments on the eyes of rabbits are mentioned.

The effectiveness of these rays in a series of 300 cases is analyzed and the following points are brought out:

(*a*) The rays exert an analgesic effect in the treatment of most superficial diseases of the eye.

(*b*) The treatments are easily given and are painless.

(*c*) The range of safety is great, and serious untoward effects were not experienced.

(*d*) Most inflammatory conditions of the cornea were benefited, especially ulcers.

(*e*) Scleritis and episcleritis usually yielded to the treatment.

(*f*) Dystrophic conditions in general were not helped.

Finally, it is contended that grenz rays are as efficacious in the treatment of superficial lesions of the eye as any other form of radiant energy and that they lack the disadvantages. It seems safe to prognosticate that the future will demonstrate their superiority.

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DISCUSSION

DR. GUSTAVE BUCKY: When I congratulate Dr. Pfeiffer on his presentation, I do not use an empty phrase, as is so often done. His work and that of his collaborators show not only the earmarks of scientific thoroughness but enter on an entirely new scientific field in this country. There are always serious obstacles in pioneer work. Dr. Pfeiffer has overcome these difficulties, for which he is to be complimented.

Dr. Pfeiffer has shown that grenz rays possess striking properties, especially fit for ophthalmologic therapy. Generally speaking, the biologic effect of grenz rays is equal to that of roentgen rays and of radium. No doubt there are differences between various therapeutic agents and radiations; in spite of the similarities of grenz rays to other electromagnetic oscillations, treatment with them can safely be called the method of choice in a number of superficial ocular conditions. My own observations, which depend greatly on the judgment of experienced ophthalmologists, mainly resemble those of Dr. Pfeiffer. There is a general consensus as to the value of grenz rays at the leading ophthalmic clinics where they were applied thoroughly, both here and abroad. I shall mention only the Presbyterian Hospital and the Knapp Memorial Hospital in New York and the eye clinics at the universities of Vienna, Berlin and Jena.

What advantages has this form of therapy over other methods? They can be enumerated as follows: 1. Grenz rays have a therapeutic effect, which has a specific character in some conditions. 2. They have an analgesic effect. 3. They are safe. 4. They are simple to handle, and the period of application is short.

Dr. Pfeiffer has covered the first two points extensively.

As to the safety of the method, clinical observations as well as experimental studies of Krasso, Erggelet and others have shown that no undesired sequelae need be expected if therapeutic and not experimental doses are applied. The production of cataract is entirely out of the question for two reasons: first, therapeutic doses are too small to damage

the lens and, second, grenz rays cannot penetrate the anterior chamber in a sufficient amount. To create slight temporary changes on the cornea, therapeutic doses must be multiplied from ten to thirty times, so that with an error of several hundred per cent in energy applied for therapeutic purposes the dose is still within the safety zone. Furthermore, as the cumulative effect of grenz rays is far less than that of roentgen rays or of radium, the treatment can be repeated until the therapeutic goal is reached. This point was especially stressed by Spiethoff, professor of dermatology of the University of Leipzig. Whereas with other forms of radiation one must stop on account of the danger of burns, the danger dose of grenz rays is so far from the therapeutic range that a cure can always be accomplished before the danger line is reached.

As low voltages are being dealt with, the apparatus is small, movable and shock proof. Its output is high, so that an ordinary treatment requires only from ten to twenty seconds, which is of great advantage in sensitive eyes and simplifies the application.

The calibration of the tubes is no longer a problem. A new device makes the measurements so simple that the physician himself is able to calibrate his machine.

As to indications, I wish to give a few examples. I consider grenz therapy the method of choice for superficial tumors of the eye and the lids. It is understood that no tumor should be treated which has penetrated to the deeper layers or has produced metastases. In general, a perfect cosmetic and therapeutic effect can be accomplished with from two to five applications of from 1,500 to 2,500 r, whereas the ordinary dose amounts to from 100 to 400 r. The treatment is repeated usually after from four to six weeks in doses over 1,000 r until the desired therapeutic effect is reached.

By applying high ray intensities, one can assume that the rays exert a destructive power on epitheliomas.

It is more difficult to understand the mechanism of the curative effect of grenz rays on hemangiomas. Relying on the destructive theory, I at first applied high intensities similar to those used on epitheliomas. Certainly I was able to obtain remarkable improvement; however, the cosmetic result was not satisfactory in that slight telangiectases developed in some of the patients. Since then I have learned to avoid these disturbing sequelae by applying small doses of not more than 300 r and repeating them not more than once in two months. Such an intensity is hardly able to create noticeable changes in the skin, either macroscopically or microscopically. In spite of this fact, hemangiomas disappear after some time. Here a biologic process of readjustment or reconstruction rather than of destruction seems to be initiated. Furthermore, telangiectases as sequelae appear only on normal skin and not where the epithelioma was originally situated, even with doses higher than 2,000 r.

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MARKED ANISOMETROPIA

REPORT OF A CASE IN WHICH FULL CORRECTION WAS ACCEPTED

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A white man aged 48, who apparently was in good health, registered in the Section on Ophthalmology of the Mayo Clinic on Sept. 16, 1936, for refraction. The glasses he had worn for the preceding four years had been broken recently, but the lenses, while they had enabled him to carry on his duties as a railroad station agent in a small community, never had provided him with satisfactory, comfortable vision. Refraction had been done many times by numerous ophthalmologists and by nonmedical refractionists. He still possessed twenty-five pairs of glasses, none of which was really useful. Poor visual acuity had been present since early childhood, but asthenopia never had been a prominent symptom. There was no history of infection or injury of the eyes, nor was there any family history of ocular disease.

The external ocular examination revealed facial asymmetry of moderate degree. There were slight flattening of the entire left side of the face and an upward twisting of the right side of the face. The position of the orbits and of the eyes, however, was essentially normal; the other structures of the anterior segments of the eyes were normal, with the exception of the corneas, which showed marked bilateral astigmatic distortion, as determined by the ring images formed by the keratoscope. This was confirmed by the use of the keratometer, which disclosed that the images of the mires were completely reversed and that there was more corneal astigmatism than could be measured by the instrument. The corneal biomicroscope revealed a bilateral, irregular, conic type of cornea, which was more marked in the right eye than in the left. There was no scarring. The epithelium and endothelium were intact. No infiltration of the substantia propria was observed, although irregularly distributed thickening and thinning of the substance were noted. The iris was normal in each eye, and no opacities of the lens were noted. Determination of the muscle balance by means of the Maddox rod and with the old correction gave results within normal limits.

Ophthalmoscopic examination did not disclose any pathologic changes in the ocular fundi; however, the details were blurred.

The results of the determination of the visual acuity prior to the actual refraction are summarized briefly in table 1. The values for visual acuity shown in this table represent the maximal values and were obtained on definite accommodative effort.

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The use of a cycloplegic was deemed unnecessary. One drop of a 4 per cent solution of cocaine hydrochloride was instilled as a mydriatic in each eye fifteen minutes prior to refraction. In table 2 are summarized briefly the results obtained with the trial case.

No presbyopic error was found. The correction was accepted readily by the patient, and he was given the full prescription as recorded and was requested to return for further observation in thirty days.

When he returned to the clinic on October 22, he said that he was enjoying for the first time in his life what he considered to be good vision. All work could be performed easily, but he was having some difficulty in reading ordinary newsprint in the evenings. On inquiry, this was found to be due to extremely poor lighting facilities for near work in his living quarters. Examination of his glasses with the lensometer revealed that the lenses had been ground properly and correctly inserted. Vision in the right eye with this correction was 6/12— and that in the left eye was 6/10+. Vision of 6/7 was obtained easily with both eyes with

TABLE 1.—*Results of Determination of Visual Acuity Prior to Actual Refraction*

Eye	Vision Without Correction	Accommodation Near Point Without Correction	Old Correction Worn at Time of Examination	Vision With Old Correction	Accommodation Near Point With Old Correction
O. D.	1/60	7 cm., 14/35, A.M.A. rating	+0.75⊂ —14.50, ax. 77	6/15	12 cm., 14/35. A.M.A. rating
O. S.	6/20—	12 cm., 14/24.5, A.M.A. rating	—1.25⊂ +7.75, ax. 13	6/12+	15 cm., 14/24.5, A.M.A. rating

TABLE 2.—*Results Obtained with the Trial Case*

Eye	Correction Accepted at Time of Examination	Vision with New Correction	A.M.A. Rating	Preferred Near Distance
O. D.	—19.25, ax. 90	6/12+	14/24.5	36 cm.
O. S.	—3.50⊂ +10.50, ax. 5	6/10+	14/24.5	36 cm.

correction, and at a distance of 36 cm., which was his preferred distance for work and for reading, he could read 14/24.5, American Medical Association rating, easily with each eye separately and with both eyes together. The temples and saddle of the frame were readjusted, but no change in the prescription was advised. The patient was asked to return for reexamination within two years.

When he was seen again on Sept. 24, 1938, he had no symptoms of any nature except for an occasional awareness of the weight of the lenses and of the slight pressure of the saddle of the frame on the root of the nose. At times he experienced a mildly annoying reflection of light from a window or from a glazed page of reading material. Vision with his correction was 6/12— in each eye and 6/7— with both eyes. For near vision he could read 14/21, American Medical Association rating, easily with the right eye and 14/28, American Medical Association rating, easily with the left eye; with both eyes, with correction, he could read 14/21, American Medical Association rating, easily and readily. Second degree fusion but not true stereopsis was obtained by testing with the Guibor fusion cards. Correct answers were obtained constantly with the Maddox four dot test. The determination of the patient's muscle balance with the red Maddox rod while he was wearing the correction revealed no hyperphoria at a distance of 6

meters, esophoria of 1 prism diopter and divergence ability of 7 prism diopters. At a distance of 33 cm. there was right hyperphoria of 0.5 prism diopter and exophoria of 4 prism diopters, and the ability to converge was excellent and well sustained. Examination with the retinoscope, keratometer, keratoscope and the corneal biomicroscope revealed no change in the appearance of the corneas since the previous examinations. Ophthalmoscopically, the ocular fundi were the same as they were at the time of the previous examinations, except for astigmatic distortion of the nerve heads. The temples and the saddle of the frame again were adjusted, but after a recheck of the refractive error by means of the trial lenses no change in the prescription was suggested. Annual reexamination was advised, and the patient was dismissed.

COMMENT

It is commonly believed that marked anisometropia and antimetropia should not be corrected fully by lenses. It might be supposed that (to employ Lancaster's term [1]) "the neuromuscular mechanism for binocular vision" could become overburdened; however, a discussion of the arguments involved in the consideration of this thesis does not lie within the scope of this report. It is desired only to draw attention to the fact that the patient accepted readily and wore in comfort a full correction for his extreme anisometropia. This is evidence that it is possible, bilaterally, to correct fully these marked errors of refraction. Eventual full correction of anisometropia in our experience, clinically, has been satisfactory.

1. Lancaster, W. B.: Aniseikonia, Arch. Ophth. **20**:907-912 (Dec.) 1938.

SIGNIFICANCE OF FALSE PROJECTION IN TREATMENT OF SQUINT

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Although there are many articles in the literature on the nature and incidence of false projection, there has been little emphasis on its importance in the treatment of squint. Pugh [1] made this general statement as to the possible effects of false projection:

> If a patient's eyes are straightened by operation while he is still using false projection, he may after operation do one of the three things: (*a*) He may, finding the visual axes parallel, develop a true projection and fuse with his eyes straight. (*b*) He may pass through a transitional stage when he learns to readjust his projection so that he eventually adapts himself to the new position of the eyes. During this transition there is a false diplopia. . . . (*c*) He may show no sign of modifying his false projection, but retain it. Such a patient suffers from a troublesome false diplopia and in his efforts to overcome this symptom he reverts more or less to his original deviation.

Travers [2] dealt with the problem as a whole but made his clinical survey from the standpoint of the effect of surgical and orthoptic treatment on false projection. The purpose of this paper is to discuss the effect of false projection on the surgical and orthoptic treatment of squint.

The phenomenon of false projection has been given a variety of names, some of which are misnomers: false macula, incongruous diplopia, abnormal retinal correspondence, abnormal binocular projection, paradoxic diplopia, false associated fixation and anomalous and false projection. Probably anomalous binocular projection is the most descriptive term. False projection is, however, somewhat simpler.

Moncrieff [3] defined false projection as "an abnormality of the sensoric relations of the two retinas which give rise to disturbances

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1. Pugh, M. A.: The Significance of False Projection in Squint, Proc. Roy. Soc. Med. **27**:1615-1622 (Oct.) 1934.

2. Travers, T. Ab.: The Comparison Between the Visual Results Obtained by Various Methods Employed for the Treatment of Concomitant Strabismus (An Essay for the Gifford Edmonds Prize in Ophthalmology), London, George Pulman & Sons, Ltd., 1936.

3. Moncrieff, W. F.: Disturbances of Visual Functions in Concomitant Convergent Squint, with Special Reference to Retinal Incongruity, Arch. Ophth. **2**: 179-197 (Aug.) 1929.

in relative localization"; Bielschowsky,[4] as "a functional adaptation of the sensory apparatus to the abnormal position of the eyes relative to a fixed object"; Pugh,[5] as "a mental reorientation of the displaced image," and Verhoeff,[6] as "a type of binocular projection in which there is quasi retinal correspondence." Worth's[7] definition is less specific but clear: ". . . the mind sometimes learns to make full allowance for the faulty position of this [the deviating] eye. So that the eccentric image, formed in the deviating eye, is mentally projected to the same spot as the true macular image, formed in the normally directed eye, and is blended with it." If to this definition it is added that the phenomenon is purely a binocular one, the definition is adequate.

False projection is frequently first discovered by the development of paradoxic diplopia after technically correct operative procedures. However, it may be revealed by clinical examination.

Bielschowsky used the method of after-images. This method in brief consists of developing after-images on each fovea by means of monocular fixation of a glowing filament presented as horizontal to one eye and as vertical to the other. The after-images should form a cross if the two foveas are coordinating. If false projection is present, the image of one filament will be displaced in a paradoxic direction. With a known distance between the patient and the screen on which the after-images are projected, the distance between the two images gives the examiner a means of calculating the angle of false projection. The contentions against this method are: 1. The test demands too much of an unintelligent patient—unwavering fixation during the ten second intervals, accurate observation and report of a fluctuating subjective phenomenon. 2. The angle of false projection depends on the angle of squint, and this angle may vary during the test. The consequent variation in the angles of projection is confusing to the patient, who may or may not report the change. 3. The test must be given in darkness or semidarkness, so the observer has few objective checks.

The best method for the determination of the presence of false projection is the use of the synoptophore. This allows a rapid and accurate measurement of the angle of squint and the interpupillary distance. It provides immediate checks on fixation, and the corneal reflexes of the patient's eyes are clearly visible to the examiner at all

4. Bielschowsky, A.: Application of the After-Image Test in the Investigation of Squint, Arch. Ophth. **17**:408-419 (March) 1937.

5. Pugh, M. A.: Squint Training, London, Oxford University Press, 1936, p. 34.

6. Verhoeff, F. H.: Anomalous Projection and Other Visual Phenomena Associated with Strabismus, Arch. Ophth. **19**:663-699 (May) 1938.

7. Worth, C.: Squint: Its Causes, Pathology and Treatment, ed. 3, Philadelphia, P. Blakiston's Sons & Co., 1906.

times. In testing for false projection the first step must be an accurate and reproducible measurement of the angle of squint. The screen test is undoubtedly the best. The only times that it is not reliable are when the vision is so low that fixation cannot be maintained even with a brilliant object and when there is a latent or a manifest nystagmus. In all other cases the screen test gives the most accurate measurement. When there is no movement of either eye under cover, the angle of deviation has been found. In testing for false projection two dissimilar test objects are placed before the patient's eyes at this angle. With normal projection or normal binocular perception the two images would be superimposed. If false projection is present, however, the two images will not be seen as superimposed but will be perceived as crossed, the image in the right eye to the left and the image in the left eye to the right. If the instrument is then adjusted so the two images approach each other or are superimposed, the angle between the two test objects will have been decreased to parallel or almost so. The patient then perceives the images as he would if the visual axes were parallel, even though the squint is still manifest. The correction of the deviation in such a patient would create for him a subjective exophoria. A slight difference (2 to 5 prism diopter) between the angle of squint as measured by the screen test and the subjective diplopia is within normal limits. The screen test measures not only the manifest squint but any phoria, which may be overcome as soon as both eyes are allowed to fix. If the discrepancy between the angle of squint as measured by the screen test and the angle of diplopia is greater than 5 prism diopters, the diagnosis of false projection is justified.

An indication of the presence of false projection can be obtained during the test for near point of convergence. In a patient with normal projection there is some point, no matter how close, where the two corneal reflexes are both centered, from which point the two eyes will diverge and converge in the normal manner. In a patient with false projection one corneal reflex will be consistently at the temporal margin of the pupil until the patient has reached his limit of convergence, when the corneal reflexes become centered, but there will be no ability to converge or diverge beyond this point.

Binocular false projection is occasionally confused with the following conditions:

1. Monocular false projection occurring in spasm or paralysis of the ocular muscles. Monocular false projection is caused by the discrepancy between the effort of the patient to move the eye and the amount of movement accomplished. As a rule, this occurs in one direction only, in the field of action of the affected muscle, and is present in the affected eye alone. Binocular false projection is dis-

closed only when both eyes are perceiving. With either eye covered, the other has perfectly normal projection in all directions.

2. Eccentric fixation or false macula. Eccentric fixation may be due to a macular defect or to other causes and in turn be associated with false projection, but it does not cause false projection nor is it synonymous with it. False macula, an extrafoveal region with visual acuity equal to that found at the fovea, rarely develops in uncomplicated squint.[8]

3. Discrepancies in projection due to inaccurate observation or report on the part of the patient. Inaccuracies do occur, but a check by the screen test will indicate whether the discrepancy is real or merely due to inattention.

4. Variable angle of squint. The angle of squint may vary and so vary the angle of diplopia, but accurate measurements show that the projection is likewise anomalous.

5. Incongruous diplopia. Duane[9] defined false projection as "paradoxic diplopia" and differentiated it from "incongruous diplopia." He considered paradoxic diplopia as opposite to what one would expect, e.g., crossed instead of homonymous, whereas incongruous diplopia presents a discrepancy between the objective and the subjective findings. Incongruous diplopia may be of the same type one would expect but of an entirely different amount. Incongruous diplopia, then, becomes paradoxic diplopia after the squint has been straightened by surgical treatment.

6. Rivalry in perception between the two eyes. It is difficult for some persons to perceive simultaneously two dissimilar images presented in such a manner that each eye sees only one image. First one image will appear, then will be succeeded by the other. This is not false projection. The images are referred to their proper place in space; but first the image of one eye is dominant and then the image of the other eye. In normal persons this can be overcome by close attention. In cases of squint this anomaly is exaggerated and becomes suppression.

7. A large negative angle gamma. In a few cases the angle between the visual axis and the optical axis is so large that the corneal reflex is located slightly to the temporal side of the center of the pupil, giving the impression of convergent squint when there is none. The presence of this anomaly is determined by the screen test, with which no deviation will be found, and by the monocular fixation test, with which the exact location of the corneal reflex can be determined.

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8. Pugh,[5] p. 23.

9. Duane, A.: Diplopia and Other Disorders of Binocular Projection, Arch. Ophth. **7**:187-210 (Feb.) 1932.

The material on which this study is based consists of data on 600 consecutive cases of strabismus taken from the files of the Orthoptic Clinic of the Wilmer Ophthalmological Institute. This number included 177 cases of uncomplicated, nonparalytic convergent strabismus without marked vertical deviation, i.e., cases in which there were normal fundi and media, good central fixation and vision permitting appreciation of diplopia and no complicating systemic or ocular disease. The term "straight" in the following tables bears this connotation: The eyes are cosmetically straight with glasses. The esophoria for distance was no more than 8 prism diopters, and the esophoria for near vision no greater than 10 prism diopters; the convergence near point was normal, the fusion amplitude was equal to normal prism convergence (24 prism diopters) and prism divergence (8 prism diopters)

TABLE 1.—*Data on Patients with False Projection* *

No. with false projection	94 (53.1%)
No. treated by surgical measures	49
No. treated by surgical measures before restoration of normal projection	30
No. treated by surgical measures after restoration of normal projection	19
No. treated by orthoptic methods and glasses	18

* The data presented in the tables are based on a study of 177 persons with uncomplicated, nonparalytic convergent strabismus.

TABLE 2.—*Data on Patients with Normal Projection*

No. with normal projection	75 (42.3%)
No. treated by surgical measures	25
No. treated by glasses alone	29
No. treated by glasses and orthoptic methods	21

power and depth perception was consistent with the vision of the two eyes. The tests were made with the refractive error corrected by glasses. In the majority of cases the refractive error was 3.0 diopters or more of hyperopia or hyperopic astigmatism. If the final result was normal binocular vision with correction of the refractive error, the result was considered successful.

In the Wilmer Ophthalmological Institute all treatment, including the surgical treatment of squint, is under the supervision of one person, the director of the institute. The conclusions drawn from this study are therefore not vitiated by lack of uniformity in treatment. Analysis of the data presented indicates that many of the failures may be attributed to lack of restoration of normal projection before operation. The eyes in only 4 of 30 cases in which surgical treatment was carried out before false projection was corrected remained straight, while those in 17 of 19 cases in which normal projection was restored before operation remained straight. The importance of false projection in the treatment of squint thus becomes apparent.

TABLE 3.—*Results for Patients with False Projection*

No. treated by surgical measures before the restoration of normal projection	30
No. whose eyes remained straight	4 (13.3%)
No. treated by surgical measures after restoration of normal projection	19
No. whose eyes remained straight	17 (89.4%)
No. treated by orthoptic methods	18
No. whose eyes remained straight	10 (55.0%)
No. advised to have operation but refused	8

TABLE 4.—*Results for Patients with Normal Projection*

No. treated by surgical measures	25
No. whose eyes remained straight	22 (88.0%)
No. treated by glasses alone	29
No. whose eyes remained straight	22 (74.7%)
No. treated by orthoptic methods and glasses	21
No. whose eyes remained straight	15 (71.4%)
No. treated by orthoptic methods with varying degrees of squint remaining after 18 months of training (results classed as failures, although there was improvement in each case)	6 (23.8%)

TABLE 5.—*Incidence of Angle of Squint in Patients with False Projection* *

Angle of Squint	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-100	Total
No. of patients with equal vision in both eyes	3	10	9	9	8	0	0	4	42
No. of patients with one eye amblyopic	5	5	10	6	8	4	2	3	43

* The angle of squint is measured in prism diopters.

TABLE 6.—*Incidence of the Angle of Squint in Patients with Normal Projection* *

Angle of Squint	0-10	11-20	21-30	31-40	41-50	51-60	61-80	81-100	Total
No. of patients with equal vision in both eyes	6	1	3	0	1	4	2	5	22
No. of patients with one eye amblyopic	9	4	7	5	0	5	1	3	34

* The angle of squint is measured in prism diopters.

TABLE 7.—*Age of Incidence of Squint in Patients with False Projection*

	1-12 Mo.	1 Yr.	2 Yr.	3 Yr.	4 Yr.	5 Yr.	6 Yr.	7 Yr.	8 Yr.	9 Yr.	Total
No. of patients with equal vision in both eyes	10	2	7	8	5	1	0	1	1	0	35
No. of patients with one eye amblyopic	13	4	11	7	4	4	1	0	0	0	44

TABLE 8.—*Age of Incidence of Squint in Patients with Normal Projection*

	1-12 Mo.	1 Yr.	2 Yr.	3 Yr.	4 Yr.	5 Yr.	6 Yr.	7 Yr.	8 Yr.	9 Yr.	Total
No. of patients with equal vision in both eyes	2	2	2	9	3	2	0	0	1	0	21
No. of patients with one eye amblyopic	4	6	7	8	5	2	3	1	0	1	37

Further analysis of the data shows that false projection occurred most often in cases of squint developing before the age of 2 years. The percentage decreased with each succeeding year thereafter.

Approximately half the patients with false projection have more or less amblyopia of one eye. The patients with amblyopia of one eye achieved normal projection, although the amblyopia was not materially decreased. The amblyopia usually accompanying squint did not interfere with the results of surgical treatment once normal projection was restored. Twenty-five patients with squint and with both amblyopia and false projection were treated surgically. The eyes of 14, in whom the amblyopia was unchanged and projection became normal, remained straight. In 11 patients in whom false projection had not been corrected, the squint returned. Amblyopia may be an accompanying factor of false projection, but it had no influence on it.

The range of the angle of squint in the majority of cases of both false and normal projection lay between 20 and 60 prism diopters. There were scattered cases on both sides of this range.

There is apparently a high age limit at which it is possible to correct false projection. An example follows:

A woman 36 years of age had had an alternating esotropia of 90 prism diopters, since the age of 3. The angle of squint was too great to allow training in normal projection until the deviation had been reduced. Recession of the right internal rectus muscle reduced the angle to 30 prism diopters. One eye was immediately occluded and training for the correction of the false projection begun. Three months later the patient had spontaneous normal projection and was admitted to the hospital for recession of the left internal rectus muscle. The eyes were then straight. After her discharge from the ward she started fusion training. At the end of six weeks her eyes were straight for both distance and near vision, with and without glasses, with normal fusion amplitude and near point of convergence. Depth perception was not quite normal but was far above monocular stereopsis. A year later the depth perception had improved, the eyes remaining otherwise unchanged.

The results in this case are interesting, especially because most adults cannot afford the time required for training. However, it is proof that false projection can, at least sometimes, be corrected as late as the age of 36.

Correction of false projection, the restoration of normal projection, is the reestablishment of the normal coordination and projection of the images received by the two foveas. There are three steps in this process: 1. The patient must become aware of the deviating eye. 2. He must be made aware of diplopia at the angle of deviation. 3. He must be trained to overcome that diplopia by superimposing foveal images. Occlusion is employed to force the patient to use the deviating eye and to become aware of it as a factor in the act of seeing. Steps 2 and 3 sometimes occur so closely together that they

can scarcely be discriminated. The patient may become aware of the foveal image of the deviating eye and at once superimpose it on the other. As a rule, however, while the patient becomes aware of the foveal image of the deviating eye, he requires much encouragement and help in seeing it in its proper relation to the foveal image of the dominant eye. Occlusion is of help at this stage. It prevents the further development of the false projection which, as an anomaly of binocular perception, probably develops as a defense mechanism against diplopia and so tends to break up the false projection by preventing its original cause.

The actual treatment for the reestablishment of normal projection is the simultaneous stimulation of both foveas. Images are presented to each fovea and manipulated in any fashion which will best attract and hold foveal fixation. Often the patient attempts to turn his eye in beyond the usual angle of squint in order to receive the image to the deviating eye on the extrafoveal area he has been accustomed to use. A rapid screening of the fixing eye will restore foveal fixation of the deviating eye.

One of the most interesting phases of the correction of false projection is the monocular diplopia that develops in some patients at one period of training. A patient will be able to see two images of the object that is being presented to one eye alone; i. e., one image received on one area of the retina will be projected in two different directions at the same time. Bielschowsky [4] explained this as a rivalry between the normal and the anomalous sensorial relations when every retinal point has two visual directions where its impressions are localized, either alternately or simultaneously. For example, a patient, aged 21, with a convergent squint of 50 prism diopters, after three weeks of training found that when he removed his glasses (which had an occluder over one lens) he could see three moons, two to the right and one to the left. This occurred only when both eyes were open; with either eye covered he saw only one moon. This phase is generally of short duration, lasting not over two weeks.

Another interesting phase is the change in size of the image perceived by the deviating eye. When the false projection is uncorrected the patient will sometimes complain that the image seen by the deviating eye is larger or smaller than that seen by the fixing eye. Whether this change in apparent size is due to the blurred outlines of the image secondary to an error of refraction or to some retinal dissimilarity has not been determined. It has been considered in its possible association with aneisokonia.

The two chief causes for failure to relieve false projection are a manifest or latent nystagmus and lack of interest and cooperation on the part of the patient.

If false projection is not corrected prior to operation for squint, one of three courses may follow: 1. If the deviation is perfectly corrected, normal projection and normal binocular vision may develop spontaneously. 2. Paradoxic diplopia may develop, which gradually disappears. 3. If it does not disappear, the deviation will return. The optimistic view is the assumption that a patient with false projection in whom perfect operative correction of the deviation is obtained will after operation have normal binocular vision. Few of them do. A patient who has early learned to associate the deviated position of one eye with the straight position of the other will subconsciously make every effort to return to that accustomed position. One interesting point in this respect is that after operation the original deviation does not return but one less than the original develops. This lesser deviation is often between 20 and 30 prism diopters, or between 12 and 17 degrees. When the eye turns in this amount the image in the deviating eye falls on the blindspot and is thus conveniently hidden without any further effort of suppression or false projection.

It is not known why false projection should occur in the presence of suppression and amblyopia or why it should occur in some cases and not in all.

The 50 per cent of patients with concomitant convergent squint having normal projection can be treated with every confidence of success by any method which seems advisable to the ophthalmologist. But the 50 per cent having false projection are in an entirely different category. If normal binocular vision after operation is to be insured, the false projection should be fully corrected before operation.

The modern consensus is that false projection is an anomaly of the cortical association of retinal images. However, the anomaly is often discussed as if it concerned only the retina. The controversy over the widely used term anomalous or abnormal retinal correspondence is therefore principally of academic interest. The clinical significance of false projection lies in its influence on the treatment of squint.

SUMMARY AND CONCLUSIONS

Approximately half the patients with convergent squint have false projection.

There is a predominance of false projection in squints occurring before the age of 2.

False projection occurs at any angle of deviation and is not influenced by the amblyopia usually accompanying squint.

Normal projection can be restored in the majority of cases.

Normal projection must be restored if any type of treatment is to result in normal binocular vision.

OCCURRENCE OF VERTICAL ANOMALIES ASSOCIATED WITH CONVERGENT AND DIVERGENT ANOMALIES

A CLINICAL STUDY

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Since an anomaly of the vertically acting muscles is often associated with an anomaly of the laterally acting muscles, a study of 11,600 persons was made in the hope of gaining some definite evidence as to the frequency of occurrence of the condition producing the vertical anomaly and the effect of one condition on the other. Of this group, 1,955 showed some anomaly of the motor muscles as judged from the norms given by Duane, and of these, 715, or 36.6 per cent, had a vertical anomaly.

Convergence excess alone or complicated with divergence insufficiency was found in 16.6 per cent, with a vertical deviation in 39.8 per cent. Divergence insufficiency alone or complicated by a secondary convergence excess was found in 2.5 per cent, with a vertical deviation in 59 per cent. Divergence excess alone or complicated by a secondary convergence insufficiency was found in 7.5 per cent, with a vertical deviation in 59.9 per cent. Convergence insufficiency with a secondary divergence excess was found in 2.8 per cent, with a vertical deviation in 40 per cent.

It was not thought practical to attempt to state the great variation in the amounts of hyperphoria and hypertropia. These varied from 1 centrad to 55 centrads.

The cause of the underaction of the vertically acting muscle could not be determined in many cases; that is, it could not be determined whether the action of the muscle was limited because of the structure, insertion or innervation of the muscle or whether the overaction was due to a primary spasm of the muscle or to a secondary deviation of the associate muscle or to a secondary contracture of the direct antagonist. Again, it is a generally accepted theory that the act of sursumvergence

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is analogous to that of convergence and of divergence, and undoubtedly some of the persons observed, especially those with the higher degrees of imbalance, had some overaction of sursumvergence.

A more or less marked underaction of the vertically acting muscles was found in the following number of patients:

Superior rectus muscle	unilateral	347
	bilateral	160
Inferior rectus muscle	unilateral	99
	bilateral	22
Superior oblique muscle	unilateral	12
	bilateral	1
Inferior oblique muscle	unilateral	17
	bilateral	3

The hyperphoria of 44 was unclassified.

Primary spasm was rare in our opinion. Other observers claim a much higher percentage of primary spasm, but this is a controversial subject. But few instances of primary spasm of sursumvergence were observed. When the function of sursumvergence was excessive, there were but few cases in which the vertical imbalance did not increase definitely in some field. This could often be demonstrated by diplopia tests but more accurately by the screen test and the screen-comitance test in the six cardinal fields. This ruled out a primary anomaly of sursumvergence in most cases.

Of the 1,955 patients studied, 873 had an uncomplicated convergence insufficiency and 20 had retraction syndromes or other similar congenital anomalies. This leaves 1,062 patients with the following anomalies:

Uncomplicated lateral deviation	347
Uncomplicated vertical deviation	358
Combined vertical and lateral deviations	357

Thus, 2 of every 3 persons in this group, or 2 of every 5 in the entire group of 1,955, had a vertical imbalance.

Of the 1,062 patients, 338 had a refractive error of 0.50 D. or less. Of these, 29 per cent had an uncomplicated lateral deviation, 47 per cent had an uncomplicated vertical deviation and 23 per cent had combined lateral and vertical deviations. Of 286 patients with hypermetropia of 2 D. or less, 36 per cent had an uncomplicated lateral deviation, 34 per cent had an uncomplicated vertical deviation and 30 per cent had combined lateral and vertical deviations. Of 205 patients with hypermetropia of more than 2 D., 50 per cent had an uncomplicated lateral deviation.

14 per cent had an uncomplicated vertical deviation and 36 per cent had combined lateral and vertical deviations. The remainder of the group of 1,062 were myopic and presented deviations which were about evenly divided among the three types.

Of the 1,062 patients, 767 had a visual acuity of 20/25 or better in each eye. Of these 28 per cent had an uncomplicated lateral deviation, 41 per cent had an uncomplicated vertical deviation and 31 per cent had combined vertical and lateral deviations. Fifty-four patients had a visual acuity of 20/25 or better in one eye and 20/40 or better in the other eye. Of these, 41 per cent had an uncomplicated lateral deviation, 16 per cent had an uncomplicated vertical deviation and 43 per cent had combined lateral and vertical deviations. Eighty-one patients had a visual acuity of 20/25 or better in one eye and less than 20/40 in the other eye. Of these, 63 per cent had an uncomplicated lateral deviation, 14 per cent had an uncomplicated vertical deviation and 23 per cent had combined lateral and vertical deviations.

Fig. 1.—R. McK., aged 2½ years, had had divergent strabismus since birth. Vision was 20/20 in each eye under atropine cycloplegia with a + 0.50 sph. There was an exotropia of 70 centrads for distance and for near vision. There was paresis of the right inferior rectus muscle. Bilateral recession of the external rectus muscle was followed by bilateral resection of the internal rectus muscle. The vertical error was not corrected. *A* shows the patient before operation; *B*, the patient after the second operation.

The test used was the screen (cover) test, combined, when practical, with the parallax test. This test was made routinely at 6 meters and 33 cm. and in the six cardinal fields at 33 to 40 cm. In cases of divergence excess and insufficiency the distance was often increased up to 150 feet (45 meters), while in cases of convergence excess and insufficiency the test object was brought in to 20 cm.

For persons with phorias and the lower degrees of tropias, the Maddox rod or the combined screen-Maddox rod test was used with the screen-parallax test to check one against the other.

Diplopia was formerly routinely plotted on the tangent curtain, but the field of its use is quite limited, since it cannot be employed for young children, for patients with suppression or amblyopia or for those having

a great amount of deviation. The aforementioned tests have been found to be much more reliable. Other tests were employed as comfirmatory when desired.

Andrew Lang has said that statistics are as a lamppost to a drunk, used for support rather than for illumination. Having no theory to support, we thought that it might be well to get what illumination we could from a review of some of the cases as representative of the various anomalies. The conclusions are drawn partly from statistics on the aforementioned group of 1,955 patients but largely from observations made on patients at the muscle clinic of the New York Post-Graduate Medical School and Hospital and of the Brooklyn Eye and Ear Hospital and on patients seen with confreres in many different cities. The data on the last three groups, of course, are not included in the statistics.

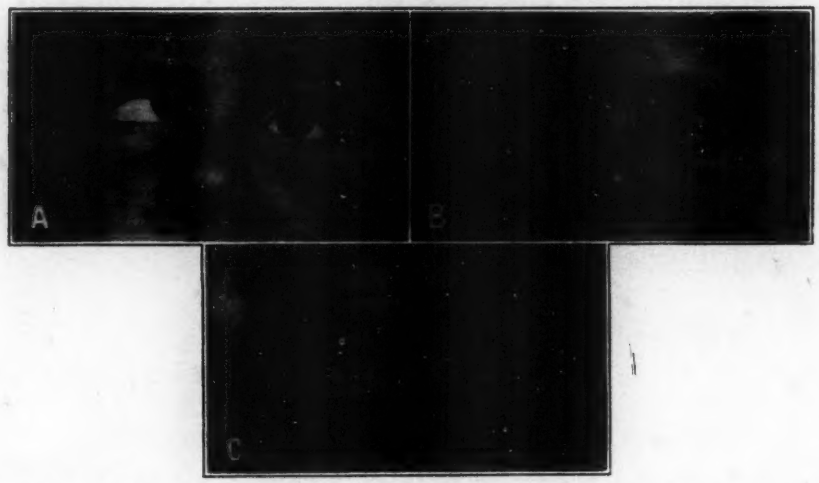

Fig. 2.—I. P., aged 6 years, had had crossed eyes since birth. There was alternating convergence strabismus, with bilateral paresis of the superior rectus muscles and bilateral secondary deviation of the inferior oblique muscles. Vision was 20/25 in each eye, with a + 1.50 D. sph. ⌒ + 0.75 cyl., axis 90 in the right eye and a + 1.25 D. sph. ⌒ + 0.50 cyl., axis 90 in the left eye. Bilateral tenotomy of the inferior oblique muscle was followed by bilateral recession of the internal rectus muscles. Later bilateral resection of the external rectus muscles was done. *A* shows the fixing left eye; *B*, the fixing right eye and *C*, the eyes after operation.

In convergent strabismus complicated with a vertical imbalance, little if any correction is obtained from plus lenses, even when the vertical error is small. Many persons with such an anomaly have been wearing low plus lenses for a number of years without improvement.

In the higher degrees of hypermetropia with squint ranging from 40 to 70 centrads, usually only from 10 to 20 centrads was corrected by lenses if a vertical imbalance were present. Two patients treated recently

at the muscle clinic of the New York Post-Graduate Medical School and Hospital illustrate this.

The first patient, a girl aged 10 years, had hypermetropia of 4.5 D. and 50 centrads of convergent squint, with no vertical imbalance. The esotropia was reduced to an esophoria of 5 centrads with full correction. The second patient, a boy of the same age, had hypermetropia of 4 D. and esotropia of 55 centrads, with a vertical imbalance. Full correction reduced the esotropia to 40 centrads, correcting but 15 centrads of the lateral deviation. These are typical examples of the effect of plus lenses in the absence or presence of a vertical anomaly. However, the unusual vertical imbalance is converted by glasses alone into a divergent squint of approximately an equal amount.

Convergence insufficiency or divergence excess, whether simple or combined, is usually made worse by the use of plus spheres. Occa-

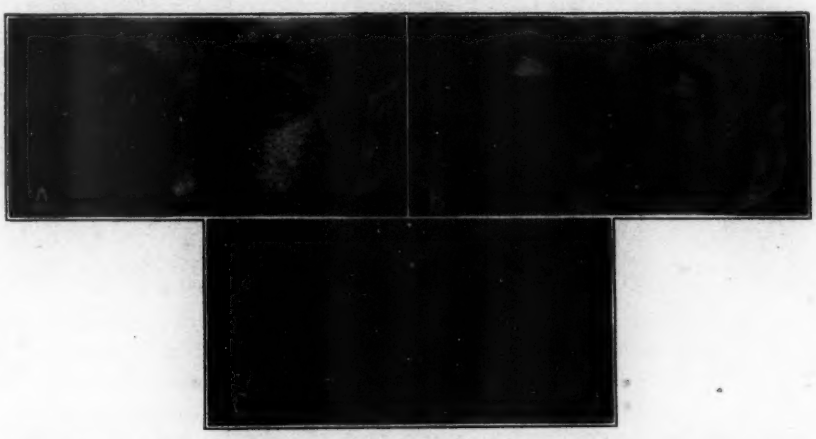

Fig. 3.—*A*, patient J. K., showing fixing right eye with the left eye diverging and higher. *B*, the patient looking up and to the right. The right eye is fixing, showing paresis of the right superior rectus muscle, with secondary deviation of the left inferior oblique muscle. *C*, the patient after recession of the left external rectus muscle and tenotomy of the inferior oblique muscle at its insertion.

sionally it is necessary to withhold both spherical and cylindric corrections unless they are necessary to relieve symptoms of headache or of asthenopia.

Pure divergence insufficiency is unaffected by the use of lenses. However, if a convergence excess is superadded, plus lenses may relieve the symptoms due to the convergence excess, while the deviation for distance remains practically unchanged.

Persons with myopia and convergence insufficiency or divergence excess are frequently helped in overcoming the lateral deviation by a full or overcorrecting myopic lens.

In all cases the vertical error should be corrected by prisms when possible. This will depend on the degree of comitancy of the vertical deviation and the degree of comfort experienced.

Orthoptic training gives varying results. Many patients will show third grade fusion during training but at all other times have a tropia. Rarely is the amount of hypertropia reduced by such training when measured by the screen test, but sursumvergence may be increased sufficiently to enable the vertical error to be overcome, at least when the subject is concentrating.

Hypertropia in the primary position may be overcome by turning or tilting of the head, and this in turn may correct the esotropia or exotropia. This result is not infrequent, and when the tilt is not marked surgical treatment may be contraindicated, and even prisms may not be well borne. However, if the tilt is disfiguring or produces symptoms, a satisfactory result can be expected by operation.

Fig. 4.—*A*, patient D. S., with a fixing left eye in the primary position. *B*, fixing left eye with the patient looking up to the left. Correction of the lateral deviation only usually results in a disfiguring right hypertropia. Tenotomy of the inferior oblique muscle may be done at the insertion before completing the resection. Recession of the internal rectus muscle may likewise be done if the relative convergence near point is not remote.

REPORT OF CASES

The following cases have been selected to show the various results seen when a vertical imbalance is present.

CASE 1.—A girl, aged 7 years, with vision of 6/6 in each eye, had hypermetropia with a + 0.25 cyl., 90 axis in each eye. In September 1934 she had right hyperphoria of 5 centrads, with no lateral deviation for distance. For near vision there was esotropia of 15 centrads with right hypertropia of 5 centrads. One year later she had right exotropia of 20 centrads with right hypertropia of 6 centrads for distance, while for near vision she had no lateral deviation and right hyperphoria of 6 centrads. She achieved fusion with depth, but orthoptic training failed to make her want to do so, except when using the instruments or when using the eyes for near vision. In September 1937 the exotropia was 27 centrads, the right hypertropia 5 centrads for distance, the exophoria 6 centrads and the right hyperphoria 5 centrads for near vision. Operation was advised.

CASE 2.—A girl, aged 9 years, had a marked divergent squint for distance. For near vision the deviation varied from an exophoria to an exotropia. Training in convergence improved the convergence near point. A recession of one external rectus muscle reduced the deviation greatly, and exercises in convergence have been continued. When the patient was seen in September 1938 she had exotropia of 20 centrads, which changed to exophoria for distance and orthophoria for near vision, with a convergence near point of 55 mm.

CASE 3.—A boy, aged 2½ years, had exotropia of 35 centrads for distance, with right hypertropia. His convergence near point was normal but poorly held. He was treated for one year without improvement. Recession of the right external rectus muscle resulted in overcorrection. This was improved by correction of the hyperopic astigmatism. Later resection of the right external rectus muscle and tenotomy of the inferior oblique muscle at the insertion gave a satisfactory result.

CASE 4.—J. S., aged 6 years, had crossed eyes at 3 years of age. Full correction of a +1.50 spherical lens had been used with no effect. The child was operated on in May 1938. The scars indicated that the right external rectus muscle had been resected and the internal rectus muscle receded. The scars looked as if the surgical technic had been excellent. I have no knowledge of the amount of squint before operation. Vision was 20/25+ in each eye, the left eye usually fixing for distance and for near vision. The screen test showed exotropia of 34 centrads for distance and 38 centrads for near vision. The relative convergence near point was 120 mm., with the left eye diverging. There was right hypertropia of 5 centrads in the primary position, which increased when the child looked up to the left. In the upper right field there was left hypertropia, owing to paresis of the right superior rectus muscle, which, however, was much less marked than the paresis of the left superior rectus muscle; hence, the predominant right hypertropia. Exercises in convergence had had little effect. Such an overcorrection is not unusual when a vertical imbalance is present.

Recession of the external rectus muscle of the diverging eye with tenotomy of the inferior oblique muscle at the insertion gave an excellent cosmetic result.

CASES 5 and 6.—A brother and sister were operated on by an excellent surgeon for convergent squint. In each instance a unilateral recession was done. One operation resulted in a divergent squint for distance and for near vision, both with and without glasses, while the other resulted in a divergent squint with glasses for both distance and near vision and without glasses for distance only. Each patient had a vertical imbalance.

CASE 7.—A man had a divergent strabismus of about 40 centrads, judged from the report received. Both external rectus muscles were receded by the attending ophthalmologist. This resulted in a convergent squint of a similar amount. The operator then advanced the left external rectus muscle, and a divergent squint of 40 centrads again resulted. There was left hypertropia of 20 centrads, owing to paralysis of the right superior rectus muscle. The vertical imbalance without doubt was responsible for the wide lateral swing of 80 centrads and back.

Space does not permit us to present more cases which give strong evidence that a vertical imbalance is an active factor in producing convergent and divergent strabismus.

If the divergence which occurs with a surgically overcorrected convergence strabismus or the convergence which occurs with an over-

corrected divergence strabismus were due to a secondary deviation caused by fixing with an eye in which a muscle had been receded or tenotomized, then divergence should be found as frequently if no vertical imbalance were present. The fact that overcorrection is practically always associated with a vertical imbalance and that the operative failures are those of many different and excellent operators would seem definite proof that the overcorrection is not due to the new adjustment of the lateral muscles but rather to the vertical imbalance.

The vertical imbalance in these cases seems definitely responsible for the unsatisfactory results from treatment by glasses, orthoptic training or operation.

SUMMARY

The following facts are significant:

With few exceptions, those persons who did not show definite improvement from lenses or from means other than operation had a vertical imbalance.

The poor results from surgical treatment, either of overcorrection or of undercorrection, in our own patients or in those observed at the various places stated consisted almost without exception of a vertical imbalance.

The operative failures of many excellent surgeons have been examined. These failures occurred in cases of both convergent and divergent squint. The choice of operation varied greatly, depending on the preference of the surgeon. Almost without exception a vertical imbalance was found to be present.

In uncomplicated convergence excess with either a lower or a higher degree of hypermetropia, a double recession will give a most satisfactory result. However, when a vertical imbalance is present, the same amount of recession for an equal amount of lateral squint may result occasionally in undercorrection but more frequently in overcorrection, about as much as the original squint. This result may be delayed or it may develop immediately after operation.

In uncomplicated divergence excess with a normal convergence near point, a conservative recession of one or of both external rectus muscles gives a good result. In the presence of a vertical imbalance, there may be an undercorrection, but much more probably a marked overcorrection.

In convergence insufficiency with secondary divergence excess, or when the divergence excess is primary, one can judge the operative results accurately when there is no vertical imbalance. But in the presence of a complicating hypertropia, the same amount of recession or resection may result in a definite overcorrection, but much more probably there will be an undercorrection of the lateral deviation.

These failures will become less frequent if the vertical imbalance can be corrected before the lateral imbalance. When the lateral imbalance is marked, it may not be possible to study the vertical imbalance properly. In such cases the lateral deviation may be corrected first, the correction of the vertical deviation being left until later, when it can be more accurately studied. Rarely the correction of the lateral deviation will lessen the vertical deviation. Not infrequently, especially in unilateral strabismus, the lateral and vertical deviations can be satisfactorily operated on at the same time. The most frequent combination, for example, is unilateral convergent or divergent strabismus associated with an overacting inferior oblique muscle of the same eye; in such instances operation should definitely be performed. When the external rectus muscle is in the grasp of the muscle forceps and has been tenotomized, the inferior oblique muscle may be tenotomized at the insertion, after which the resection or recession of the external rectus muscle is continued. For lesser amounts of overaction of the inferior oblique muscle, a procedure such as that of Blaskovics should be considered. The inferior oblique muscle was receded in 1 case with satisfactory results.

CONCLUSIONS

1. Vertical anomalies are frequent complications of lateral deviations.

2. No person with lateral strabismus should be operated on or otherwise treated without a thorough study and analysis of the vertical element, when present.

3. When one is operating to correct a lateral deviation, it must be remembered that the rules are not the same if a vertical factor is present.

4. In cases of convergent strabismus in which it is thought best not to correct the vertical deviation first, we believe that there is less danger of an overcorrection if the first procedure is resection of one or of both external rectus muscles, recession of the internal rectus muscles being left for a finer adjustment later if desired.

5. The more nearly one evaluates the several elements in these complicated cases, the greater will be the percentage of success.

DISCUSSION

DR. ALBERT C. SNELL, Rochester, N. Y.: I have not compiled any great amount of data, but in a study of the muscle anomalies in over a thousand cases of myopia, which I reported in the 1936 *Transactions of the Section on Ophthalmology of the American Medical Association* (page 49), I found that convergence insufficiency was rare, its frequency being 4 per cent (that is, confining my statistics entirely to cases of myopia); that hyperphoria associated either with exophoria or with esophoria had an incidence of about 5 per cent, and that divergence

excess and esotropia were common. However, the last condition was usually found associated with abnormal visual acuity, at least in one eye. In other words, in these cases of divergence excess in myopia I felt that the myopia was probably not the cause for the divergence excess, because I have records of a number of patients who ordinarily were far sighted and had a divergence excess and became myopic.

One constantly sees, of course, some patients with low degrees of myopia who have persistent convergent strabismus, but it is still more common to find divergence with myopia. But as I said, it seems that the relation of cause and effect in this class of cases has not yet been established. I am under the impression that divergent excess is probably present in a large percentage of cases of progressive myopia.

In the cases of myopia I found convergence insufficiency in about 1 to 4 per cent and a subnormal amplitude present in only 1 per cent. In a large proportion of all cases of lateral deviation factors other than ametropia are present, for example, anisometropia, convergence and divergence excess, vertical deviation and defective fusion sense. In a recent review of a limited number of cases convergence insufficiency was found associated with hypermetropia much more often than with myopia, the ratio being 1:8. Convergence excess is more often found associated with myopia than with hypermetropia, the ratio being 1:3.

I am in agreement with Dr. White and Dr. Brown that in a large number of cases exophoria begins as a convergence insufficiency, especially when a vertical deviation is present. When there is present a vertical deviation of 8 prism degrees or more associated with a divergence, an operation to correct this deviation is usually indicated and as a general rule should be performed at an early date. I am, of course, entirely in agreement with Dr. White and Dr. Brown that when the vertical deviation is combined with phorias or tropias, the handling of the vertical deviation is extremely important.

Dr. P. Chalmers Jameson, Brooklyn: As I see it, the theme of the paper presented is the erratic return of correction. The authors have dwelt on the computation, as Dr. Snell so ably did later. Computation is an important matter if one is going to do work on the muscles, and it has been considered much more in recent days than formerly. One used to estimate the deviation alone and then operate and trust to luck, but that is not the case now.

In cases in which there are no vertical deviations, there is nearly always an erratic return of correction of 8 or 10 per cent. The ophthalmologist who states that he always gets a simon-pure correction has forgotten something. He usually has some operation of his own, some devisement of his own, which he wants to emphasize.

The muscles are large at times. I dwell on the condition of the muscles because it is so easy to obtain information in that way. The point that I want to make is that in coming to a conclusion as to how one is going to avoid these erratic results, one ought to obtain the information by exposure of the anatomic field. I always speak of an exposed tenotomy. The exposed tenotomy should always be done; in addition to the muscular condition, one can learn a good deal about the facial conditions.

Dr. White and Dr. Brown have talked about the retraction syndromes, the facial conditions. There are many modifications of facial conditions. After exposure, one can pick up a capsule and ascertain its tension, and one either can do a vertical capsulotomy if the tension is great or can leave it alone. After exposure, one can find whether a muscle is very large, such as one of those congenital, enlarged muscles, or whether it is attenuated; and often if it is attenuated, since a deviation does not always occur on a nonattenuated side, one can close the wound and operate on the other side, thus avoiding the erratic return of correction which so frequently occurs.

PARALYSIS OF THE EXTRAOCULAR MUSCLES

CLINICOANATOMIC CONSIDERATIONS; REPORT OF CASES OF PARALYSIS OF THE OCULOMOTOR AND ABDUCENS NERVES DUE TO UNUSUAL CAUSES

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The anatomic and etiologic diagnosis of paralysis of the oculomotor, trochlear and abducens nerves is usually not difficult. A thorough knowledge of the course, and especially of the relationships, of the three nerves and of the predilections of certain infectious, toxic, vascular and neoplastic processes for these structures helps in clinical orientation. Frequently these nerves are implicated by diseases and injuries of the structures with which they are anatomically related, and a knowledge of the anatomic and pathologic pictures of, and the clinical manifestations produced by, the implicated structures is indispensable for the diagnosis of palsy of the extraocular muscles.

The most common causes of paralysis of the extraocular muscles still remain syphilis, vascular lesions, diabetes, neoplastic processes, acute suppurative and nonsuppurative infections of the nervous system, chronic degenerative processes, exogenous intoxications, mushroom poisoning, botulism and myasthenia gravis. A diagnosis of some of these diseases is possible after a single examination. For others, a somewhat prolonged period of observation with the aid of roentgen examination and other special procedures is necessary in establishing an anatomic and etiologic diagnosis. In addition to complete ophthalmoscopic and neurologic studies, a detailed roentgen study of the skull, vestibular studies, spinal fluid studies and at times encephalographic studies of the ventriculosubarachnoid system may be required as aids for an accurate diagnosis. In certain instances, only a period of observation can help in establishing a diagnosis. Thus, paralysis of the abducens nerve may be the sole evidence of an intrapontile neoplasm for weeks or months, only to be followed by manifestations pointing to the implication of other structures of the brain stem. The same holds true for slowly developing tumors in the interbrain and midbrain, where only the development of additional symptoms covering a period of many

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months can establish a definite diagnosis. In recent years the use of prostigmine as a therapeutic test has proved to be of definite benefit in establishing a diagnosis of myasthenia gravis when the extraocular muscles are the only ones involved.

The purpose of this presentation is to report some experiences with involvement of the extraocular muscles due to unusual causes.

PARALYSIS OF THE OCULOMOTOR NERVE

While it is conceded by the majority of ophthalmologists and neurologists that the most common cause of retrobulbar neuritis is multiple sclerosis, it is likewise agreed that in some cases retrobulbar neuritis is due to diseases of the paranasal sinuses, especially of the sphenoid and ethmoid sinuses. It is not generally known, however, that the nerves supplying the extraocular muscles may be implicated by the infection of these sinuses. When one considers the close relationship between the cranio-orbital nerves in their course from the posterior fossa to the sphenoid fissure and through the orbit, it is surprising that diseases of the paranasal sinuses do not involve these nerves more frequently. The extensive anatomic investigations of Onodi,[1] Sluder,[2] Schaeffer[3] and others have demonstrated that by reason of extensive pneumatization of the nasal sinuses the oculomotor and other cranio-orbital nerves are frequently separated from the ethmoid sinus and, more commonly, from the sphenoid sinuses by only thin bone and that actual dehiscences in relation to these structures are not rare. Considering the frequency of infection in the posterior sinuses, one would expect frequent involvement of the oculomotor and other cranio-orbital nerves from rhinogenic causes. Yet Onodi[1] reported only a few isolated cases, and a review of the more recent literature failed to disclose any cases. In his report Onodi[1] referred to Trantas, who observed complete ophthalmoplegia in suppuration of the ethmoid and sphenoid sinuses associated with pain referable to the trigeminus nerve. Two cases of this type have been observed and are worthy of report.

CASE 1.—H. C., a man aged 70, had quinsy on the left side in February 1938. The abscess ruptured spontaneously, and the patient was in good health until the middle of March 1938, when he noticed some pain in the region of the left upper incisor, radiating to the back of the head and to the left eye. Some days later swelling and fulness of the left eye were observed, followed by ptosis of the left upper eyelid. There were no other significant symptoms.

1. Onodi. L.: The Rhinogenic and Otogenic Lesions of the Third, Fourth, Fifth and Sixth Cranial Nerves, J. Laryng., Rhin. & Otol. **14**:304 and 360, 1914.

2. Sluder, G.: Concerning Some Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1918.

3. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal, Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

On the patient's admission to the Graduate Hospital of the University of Pennsylvania on April 20 examination showed complete paralysis of the third, fourth and sixth cranial nerves on the left side. Roentgen studies revealed bilateral ethmoiditis and an infection of the maxillary sinuses, more marked on the left than on the right, and some infection of the left premolar teeth on the left side. All other laboratory studies, as well as somatic and neurologic examinations, gave negative results. The teeth were removed without any appreciable change in the condition. March 30 a submucous resection was performed, and satisfactory drainage of the sinuses was procured. Within a few days the pain disappeared, and this was followed by a nearly complete recovery of the muscles supplied by the third, fourth and sixth cranial nerves.

CASE 2.—L. J., a woman aged 40, with an irrelevant family history and past medical history, had a cold in the head early in October 1937, and a few days later a gnawing pain developed over the right eye. On October 13, after a displacement treatment in the nose, the patient's upper eyelid drooped, and whenever she lifted it with her fingers, double vision developed. About this time severe pain developed over the right side of the forehead, in the eyeball and in inner canthus and at times over the right side of the head.

On the patient's admission to the Graduate Hospital of the University of Pennsylvania on October 19 a thorough physical examination disclosed only a complete internal and external ophthalmoplegia of the right third nerve. Routine studies of the blood and a detailed study of the spinal fluid gave negative results. Roentgenograms of the skull were normal; roentgenograms of the sinuses showed evidences of sinusitis involving the right ethmoid, the right maxillary and the left ethmoid sinuses.

Dr. G. M. Coates first treated the patient by shrinking the nasal mucous membranes. While this relieved the pain in the head, the paralysis persisted. He then opened the sinuses but found no free pus. On discharge from the hospital on October 30 the patient still had some pain and complete paralysis of the third nerve. In the succeeding few months, however, the paralysis cleared almost completely.

Comment.—As previously stated, the involvement of the extraocular muscles in association with sinusitis is relatively rare despite the frequent close anatomic relationship. Grove [4] stated that ocular paralysis sometimes follows operations on the ethmoid and sphenoid sinuses; that the trochlear and oculomotor nerves are usually involved, and that recovery is the rule. He reported a case in which operation on the antrum was followed by paralysis of the external rectus and inferior oblique muscles, without recovery after two and a half years. Immediately after the operation there was proptosis of the eye and ecchymosis.

In both cases herein reported there was, in addition to palsy of the extraocular muscles, considerable pain in the distribution of both the first and the second division of the trigeminus nerve. It is reasonable to assume that in case 1 the involvement of the cranio-orbital nerves was due to invasion of these nerves by reason of the proximity of the infected sinuses, as suggested by Sluder [2] and Onodi.[1] Surgical drainage

4. Grove, R. C.: Unusual Combination of Ocular Paralysis Following Radical Operation on the Antrum, Arch. Otolaryng. **27**:275 (March) 1938.

resulted in an immediate improvement in this case. On the other hand, in case 2 a better explanation is that a toxic neuritis existed, i. e., the infection traveled from the infected mucous membranes along the nerves and recovery was therefore slower, as is usually the case in toxic neuritis. Localized meningitis over the greater wing of the sphenoid bone in the middle fossa is a possible but not a probable explanation.

Pain referable to the trigeminus nerve in association with palsy of the extraocular muscles occurs more commonly in a variety of conditions other than sinusitis. Basal meningitis, especially of syphilitic origin, tumors and injuries at the base of the brain, thrombosis of the cavernous sinus, aneurysms in relation to the cavernous sinus and infections and neoplasms of the orbit are some of the more common conditions. Herpes ophthalmicus may be followed by palsy of the extraocular muscles, as in the case of Ironside,[5] who reported herpes ophthalmicus on the left side with corneal ulceration, followed a few days later by complete paralysis of the pupil and five weeks later by paresis on the right side. He ascribed the involvement of the third nerve to meningeal irritation predisposing to such involvement and the hemiparesis to meningeal irritation predisposing to thrombosis. Recovery did not take place in Ironside's case after two and a half months. On the other hand, Nicolau and Drăganescu [6] reported a case of ophthalmic herpes zoster and total paralysis of the oculomotor nerve, with marked recovery. In their case, four days after onset of the pain there developed mydriasis with loss of the light reflex and ptosis. Seven days after the onset of pain there was complete paralysis of the third nerve, followed in a few days by keratitis with cyclitis; regression began only after five weeks, and three months after the onset of the illness slight ptosis of the left lid, slowness of the pupillary reaction to light and residual keratitis were still present. The authors expressed the belief that in their case the virus of herpes zoster traveled along the frontal nerve to the gasserian ganglion, thence to the nasal nerve, the ophthalmic ganglion, the long ciliary nerves and the third nerve via the motor route of this nerve, to the ophthalmic ganglion.

A consideration of the 2 cases reported here focuses attention on the fact that the occurrence of pain in the first and second divisions of the fifth nerve followed by palsy of the extraocular muscles may be due to the extension of the infection by continuity or it may be indirect along the nerves, as suggested by Nicolau and Drăganescu in their case

5. Ironside, R.: Case of Left-Sided Herpes Zoster Ophthalmicus, with Incomplete Left Oculomotor Palsy and Contralateral Hemiplegia, Tr. M. Soc. London **56**:45, 1933.

6. Nicolau, P., and Drăganescu, S.: Zona zoster ophtalmique et paralysie totale de l'oculo-moteur commun, Rev. d'oto-neuro-opht. **14**:479 (July-Aug.) 1936.

of herpes zoster ophthalmicus. What is probably more important is the fact that the sinus infection need not be severe, as is borne out by my case 2 in which it is more likely that the infection traveled along the nerves.

PARALYSIS OF THE ABDUCENS NERVE

The abducens nerve is one of the most commonly affected cranial nerves and has received considerable attention on the part of the ophthalmologist, the otolaryngologist and the neurologist. It has a long and complicated course, with many possibilities for mechanical compression, stretching and infection from adjacent structures, which are commonly the seat of infections. Valuable contributions to the understanding of the various pathologic processes of this nerve have been made by the anatomic, pathologic and clinical investigations by Onodi,[1] Dorello,[7] Eagleton,[8] Houser[9] and others. The role of Dorello's canal, which is formed by the posterior clinoid process, the tip of the petrosa and Gruber's ligament, cannot be overestimated, not only in infections of the temporal bone but, as was pointed out by Houser, in infections of the paranasal sinuses and, as will be pointed out in this presentation, in other conditions, such as increased intracranial pressure, spontaneous subarachnoid hemorrhage and trauma.

It has been estimated that paralysis of the abducens nerve occurs two or three times more often than palsy of other isolated oculo-orbital nerves. The third nerve is next in order of frequency, and the fourth nerve is least often separately involved (Sears[10]).

In the majority of cases the causes of unilateral paralysis of the abducens nerve are easy to find. It is well known that unilateral and even bilateral paralysis of the external ocular muscles, in the presence of increased intracranial pressure due to tumors, has no localizing value. The most likely explanation for the occurrence of paralysis of the abducens nerve in states of increased intracranial pressure is the stretching of this nerve by the dura, with which it is intimately associated in a large part of its course. When one bears in mind the fact that this nerve has to ascend on the clivus, then make a sharp turn to enter the

7. Dorello, P.: Ueber die Ursache der transitärischen Abduzenslähmung bei Mittelohreiterungen, Internat. Centralbl. f. Ohrenh. **4**:418, 1906.

8. Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386 (March) 1930.

9. Houser, K. M.: Anatomic Relation of the Sphenoid Sinus to Dorello's Canal: Abducens Paralysis, Arch. Otolaryng. **16**:488 (Oct.) 1932.

10. Sears, W. H.: Isolated Paralysis of External Rectus in Acute Suppurative Otitis Media, Pennsylvania M. J. **13**:844, 1910; Otogenic Paralysis of the Abducens, with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion, Ann. Otol., Rhin. & Laryng. **35**:348 (June) 1926.

rigid Dorello canal, and when one further remembers that the dura is held taut by the tentorium and the falx cerebri, it is perhaps not difficult to understand why this nerve is stretched by ventricular dilatation acting on a rigid framework. In tumors of the brain early paralysis of the sixth nerve may be of value diagnostically, especially in intrapontile lesions, and perhaps in lesions of the cerebellopontile angle. I observed 1 patient in whom palsy of the external rectus muscle was the first symptom of a pituitary tumor. In this case there were no other ocular disturbances, although the patient had an enormously enlarged sella turcica with the deformation characteristic of an intrasellar tumor.

The common occurrence of paralysis of the abducens nerve in association with disease of the temporal bone is well known. It is of sufficient importance, however, to point out that the Gradenigo syndrome (palsy of the abducens nerve, pain in the face and otologic infections) may no longer be regarded without apprehension, since this syndrome is the forerunner of the more grave apical petrositis with its intracranial complications (Yaskin and Kornblum [11]).

The following are the more common causes of paralysis of the abducens nerve: intrapontile tumors and vascular lesions; meningitis, tumors, abscesses, injuries and vascular lesions at the base of the brain; localized meningitis (Gradenigo's syndrome) at the petrous apex and petrositis; lesions of the cavernous sinus and orbit; increased intracranial pressure and myasthenia gravis. Paralysis of the abducens nerve is occasionally observed after spinal anesthesia. A satisfactory explanation has not been offered for this occurrence.

Paralysis of the abducens nerve may occur secondary to involvement of the sphenoid and ethmoid sinuses. Relatively few such cases are recorded (Onodi,[1] Sluder [2] and Diamant [12]). The explanation given for the occurrence of the paralysis in these cases is the proximity of the inner wall of the sinuses to the abducens nerve. Houser [9] has enriched the knowledge on the subject by pointing out that the abducens nerve is most vulnerable in the region of Dorello's canal, where, in a great many instances, the interior of the sphenoid sinuses is separated from the canal by thin bone, so that inflammation of the sphenoid sinuses may easily cause inflammatory reactions in Dorello's canal. Occasionally one observes contralateral paralysis of the abducens nerve in diseases of the ear and of the sinuses. Sears [10] presented the following interesting possibilities for this condition: (*a*) unrecognized aural disease on the side of the paralysis; (*b*) coincident contralateral disease of the sphenoid

11. Yaskin, J. C., and Kornblum, K.: Neurologic Aspects of Petrositis, Arch. Neurol. & Psychiat. **37**:307 (Feb.) 1937.

12. Diamant, M.: Les parésies isolées du nerf moteur oculaire externe, Acta oto-laryng. **25**:550, 1937.

sinus; (*c*) homolateral disease of an asymmetric sphenoid sinus, which is in direct contact with the sixth nerve on the opposite side (Onodi [1]), and (*d*) pressure by the basilar artery or its branches associated with meningitis serosa or cerebral edema (Cushing [13]).

The injection of alcohol into the sphenopalatine ganglion for the relief of Sluder's neuralgia is occasionally followed by transient homolateral paralysis of the abducens nerve (Sluder [2]). Shuster [14] had 2 patients with this type of paralysis. The condition in 1 case followed injection into the ganglion by the nasal route through the sphenopalatine foramen, while that in the other case followed injection into the ganglion by the posterior foramen route. Both of Shuster's patients recovered within three months. No satisfactory explanation has been offered for this paralysis. Sluder [2] explained the occurrence of the paralysis by the presence of loose tissue in the sphenomaxillary fossa, so that the injected alcohol passed outward and upward, reached the abducens nerve in the sphenoid fissure and paralyzed the nerve.

In this presentation attention is directed to a number of cases of (1) isolated unilateral paralysis of the abducens nerve of unknown origin and to some unusual cases of (2) isolated bilateral paralysis of the abducens nerve.

Isolated Unilateral Paralysis of the Abducens Nerve of Obscure Origin, Terminating in Recovery.—Nine cases of isolated unilateral paralysis of the abducens nerve without any definite causation and followed by complete recovery were observed in the past twelve years. Three of the patients were females from 26 to 54 years of age; 6 were males from 6 to 68 years of age. None of the patients gave any antecedent history of acute infection or trauma, nor was there any evidence of syphilis, other chronic infections, intoxications or diabetes. Complete studies in most of the cases failed to reveal any anatomic or chemical causes for the paralysis, and spontaneous recovery took place in from two weeks to several months.

No satisfactory cause is offered for the paralysis of the external rectus muscle in these cases. The condition would seem to bear a strong analogy to Bell's palsy, the cause of which is likewise obscure. In some instances it may be due to neuritis caused by "exposure," while in others it may be of the nuclear type, possibly due to a virus infection. Just as the palsy of the facial nerve in Bell's palsy is blamed by some investigators on the bony canal in the temporal bone, so one may conceive a lesion in Dorello's canal in these cases of palsy of the abducens nerve of obscure origin.

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13. Cushing, H.: Strangulation of the Nerve Abducens by Lateral Branches of the Basilar Artery in Cases of Brain Tumor, Brain **33**:204, 1910.

14. Shuster, B.: Personal communication to the author.

Isolated Bilateral Paralysis of the Abducens Nerve.—Bilateral involvement of the abducens nerve is occasionally observed in the following conditions:

(*a*) An intramedullary lesion, with associated implication of the seventh nerve and of the long sensory and motor pathways.

The etiologic factor in these cases is neoplastic or inflammatory. The onset is usually gradual.

(*b*) Syphilitic basal meningitis.

Uhthoff [15] stated that in 150 cases of neurosyphilis paralysis of the abducens nerve occurred 27 times and bilateral paralysis of the abducens nerve 6 times. Bilateral paralysis of the abducens nerve of syphilitic origin has also been reported by Roger [16] and others.

(*c*) Intoxication due to barbiturates, as reported by Palmer,[17] and in association with diabetes, as reported by Villard, Viallefont and Bouziques.[18]

(*d*) Increased intracranial pressure as a late symptom of tumor of the brain.[19]

(*e*) Facial diplegia, with which paralysis of the abducens nerve has been reported by a number of authors to be associated in a congenital syndrome, characterized by bilateral clubfoot, bilateral palsy of the facial nerve and bilateral paralysis of the abducens nerve.[20]

(*f*) Lymphatic leukemia.[21]

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15. Uhthoff, cited by Nonne, M.: Syphilis and the Nervous System, translated by C. R. Ball, Philadelphia, J. B. Lippincott Company, 1913, p. 135.

16. Roger, H.: Paralysie bilatérale du VI d'origine syphilitique, Rev. d'oto-neuro-opht. **10**:520 (July-Aug.) 1932.

17. Palmer, G. T.: Polyneuritis Following Epivan Anesthesia: Report of a Case, New Zealand M. J. **35**:21 (Feb.) 1936.

18. Villard, H.; Viallefont, H., and Bouziques: Double paralysie du moteur oculaire externe chez un diabétique, Rev. d'oto-neuro-opht. **12**:425 (June) 1934.

19. Spiller, W. G.: Bilateral Paralysis of the Facial and Abducent Nerves as a Remote Effect of Tumor of the Brain, Ann. Surg. **101**:329 (Jan.) 1935.

20. Alajouanine, T.; Huc, G., and Gopcevitch, M.: Quatre cas d'une affection congénitale caractérisée par un double pied bot, une double paralysie faciale et une double paralysie de la sixième paire, Rev. neurol. **2**:501 (Nov.) 1930. Decourt, J., and Perreau, P.: Syndrome congénitale caractérisé par une double paralysie faciale, une double paralysie de la sixième paire et un double pied bot, Arch. de méd. d. enf. **38**:419 (July) 1935. Phillips, W. H.; Dirion, J. K., and Graves, G. O.: Congenital Bilateral Palsy of the Abducens, Arch. Ophth. **8**:355 (Sept.) 1932. Roger, H.; Aubaret, and Reboul-Lachaux, J.: Paralysie des deux VI avec exophthalmie bilatérale, Rev. d'oto-neuro-ocul. **5**:36 (Jan.) 1927. Gareiso, A., and Barbieri, C. A.: Sobre un caso de parálisis congénita bilateral del facial y motor ocular externo, con pie bot doble y congénito, Prensa méd. argent. **20**: 1713 (Aug. 2) 1933.

21. Howell, A., and Gough, J.: Acute Lymphatic Leukaemia with Facial Diplegia and Double Abducens Palsy, Lancet **1**:723 (April 2) 1932.

(*g*) The bilateral Gradenigo syndrome[22] and sinus thrombophlebitis.[23]

(*h*) Cranial trauma.[24]

A review of the literature reveals that isolated bilateral palsy of the abducens nerve is usually of gradual onset and is frequently accompanied by other signs of focal lesions in the brain. The observation of 2 patients in whom the onset of the palsy was rapid and limited to the abducens nerve prompted the following report:

CASE 3.—H. D. W., a widow aged 51, was admitted to the Graduate Hospital of the University of Pennsylvania on March 12, 1936, and was discharged on April 2. The family history and the past medical history were irrelevant except that several members on the paternal side had deafness and the patient had been deaf since late adolescence. In addition, she had undergone appendectomy, tonsillectomy and thyroidectomy. On Jan. 22, 1936, the car which she was driving skidded into a pole. She did not remember exactly what happened, but evidently she struck her face against the steering wheel. When she regained consciousness, after a short period, she had a tooth and part of the steering wheel in her hands. When she arrived at the hospital examination revealed a fracture of the maxilla, loss of three teeth and complete internal convergent strabismus. A roentgenogram of the skull failed to reveal any fracture other than that of the maxilla. The spinal fluid was not under increased pressure and was clear and normal in every detail.

Examination at the Graduate Hospital of the University of Pennsylvania revealed no significant abnormalities except complete bilateral paralysis of the external rectus muscle and nearly complete bilateral deafness. A second roentgen examination failed to reveal evidence of fracture of the skull. The results of all other laboratory tests were negative.

The patient did not improve during her residence in the hospital and when last examined still had complete bilateral paralysis of the abducens nerve.

CASE 4.—D. W. P., aged 58, single, a tinsmith, was admitted to the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases on March 21, 1936, complaining of headache, stiffness of the neck and inward turning of the eyes. The family history and the past medical history were irrelevant. On March 5 the patient awoke with rigidity of the neck and pain in the head. On March 7 both eyes turned inward, and he had persistent diplopia. He made no other significant complaints. He was well nourished, with normal pulse and temperature;

22. Malan, A.: Syndrome de Gradenigo bilatérale: De l'importance de l'examen radiologique des pyramides pétreuses dans ce syndrome, Ann. d'oto-laryng., February 1931, p. 129.

23. Greenfield, S. D.: Etiology and Pathology of Paralysis of the Abducens Nerve Associated with Sinus Thrombophlebitis, Arch. Otolaryng. **19**:336 (March) 1934.

24. Loepp, W.: Röntgenbefund bei traumatischer Abduzenslähmung, Röntgenpraxis **7**:325 (May) 1935. Dupuy-Dutemps, P.: Paralysie traumatique des deux nerfs de la sixième paire, Bull. et mém. Soc. franç. d'opht. **48**:444, 1935. Rollet, J.: Recherches sur la pathogénie de la paralysie bilatérale du nerf moteur oculaire externe, d'origine traumatique, Bull. Soc. d'opht. de Paris, January 1933, p. 65; Névrotomie optico-ciliaire: Résultats éloignés, Lyon méd. **141**:563 (May 13) 1928.

the blood pressure was 178 systolic and 106 diastolic; there was an apical systolic murmur and moderate arteriosclerosis. The mental condition was clear. Neurologic abnormalities included moderate angiosclerosis of the retinal vessels, complete bilateral paralysis of the external rectus muscles and moderate rigidity of the neck, without a Kernig or a Brudzinski sign.

Lumbar puncture revealed low pressure of the spinal fluid, which was xanthochromic and thick. It was necessary to perform aspiration in order to obtain a specimen. The spinal fluid contained 44 erythrocytes and 152 mg. of protein per hundred cubic centimeters; the Pandy reaction was plus 4, and the colloidal gold curve, 0121111000. Fehling's solution was reduced, and the Wassermann reaction was negative. The Wassermann reaction of the blood was also negative. Roentgen examination of the skull revealed a rather elongated and deep sella turcica, measuring 1.8 by 1.2 cm. in depth; there was no evidence of erosion or of increased intracranial pressure.

The patient's condition gradually improved; the blood pressure dropped, so that on May 9 it was 136 systolic and 86 diastolic. Examination of the spinal fluid on April 7 showed 12 cells, a total protein content of 57 mg. per hundred cubic centimeters and a colloidal gold curve of 0011111000. On May 4 there was nearly full rotation of the left eye but some weakness of the right external rectus muscle. When the patient was last examined, on September 15, there was complete recovery of both external rectus muscles.

Comment.—In consideration of the origin, course and relationship of the abducens nerve, the sudden and isolated bilateral involvement of the nerve in these 2 cases can best be explained by assuming that the lesion was in Dorello's canal. It is difficult to conceive of a bilateral lesion within the pons, the cerebelloptontile angle or the cavernous sinus which is not associated with many other symptoms. In Dorello's canal the abducens nerve is nearly isolated and can be involved without implicating any other structures. Furthermore, the structure of Dorello's canal is such that it can easily implicate the abducens nerve. The researches of Dorello,[7] Eagleton [8] and others [25] have shown that this canal is frequently anomalous and, when unusually narrow or short, may be the cause of paralysis of the abducens nerve. In case 1 the impact to the maxilla may have caused concussion, resulting in immediate compression of the two nerves. In case 2 the subarachnoid hemorrhage may have caused sufficient secondary meningitis to produce bilateral compression of the nerve within this canal. It is interesting to note that of the numerous cases of spontaneous subarachnoid hemorrhage this is the first in which there was isolated bilateral paralysis of the sixth nerve.

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25. Vail, H. H.: Anatomical Studies of Dorello's Canal, Laryngoscope **32**: 569 (Aug.) 1922. Wheeler, J. M.: Paralysis of the Sixth Cranial Nerve Associated with Otitis Media, J. A. M. A. **11**:1718 (Nov. 23) 1918. Sjoberg, A. A.: Contribution to Knowledge of the Genesis of Certain Symptoms of Apicitis. Acta oto-laryng. **19**:479, 1934.

SUMMARY

A clinicoanatomic review of the cranial-orbital nerves is given. The relation of the paranasal sinuses to the nerves supplying the extraocular muscles is stressed.

The role of Dorello's canal as a cause of paralysis of the abducens nerve in infections in the region of the petrous apex, in infections of the sphenoid sinus, in increased intracranial pressure and in injuries and vascular lesions at the base of the skull is discussed.

The causes of the paralysis of the various extraocular muscles is discussed.

Two cases of paralysis of the extraocular muscles with pain referable to the trigeminus nerve and an associated sinus infection are reported.

Nine cases of isolated unilateral paralysis of the abducens nerve of obscure origin and terminating in recovery are reported.

The causes of bilateral paralysis of the abducens nerve are discussed, and 2 cases of isolated bilateral paralysis of the abducens nerve are reported.

CONTACT GLASSES FOR CORRECTION OF REFRACTIVE ERRORS IN MONOCULAR APHAKIA

PRODUCTION OF BINOCULAR SINGLE VISION

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The problem of refraction in cases of monocular aphakia is as old as ophthalmology itself. The difficulty in prescribing lenses for both eyes is frequently encountered, and the explanation given as to why the two eyes cannot function harmoniously is usually hard for the patient to accept.

Heretofore when a patient presented himself with a monocular cataract it was necessary to decide whether to operate or not. The decrease in the visual field and the annoyance of an economically blind eye were points in favor of operation. However, the disappointment to the patient when the refractive errors of the two eyes could not be harmoniously corrected was usually the deciding factor against operation. Only since the use of contact glasses has this factor been overcome.

HISTORY OF CONTACT GLASSES

Contact glasses were first used by Herschel in 1827, although the first experiments in the departure from the ordinary optical lens dates back to 1669, when Wren devised a hyperbolic glass for use in cases of conical cornea. Herschel's contact glasses were made by casting from gelatin molds. Since Herschel's time, numerous ophthalmologists have used contact glasses with marked success, the increase in their use paralleling the improvements in the manufactured product. Some of these lenses are blown to specifications, others are ground, and still others are a combination of the two.

PRACTICAL APPLICATIONS

Besides the cosmetic value of contact glasses, their usefulness has been definitely proved in cases of keratoconus in which they have formed the best method of correction, eliminating the need for operation.

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In all conditions of ametropia they have proved valuable. The fluid meniscus between the cornea and the contact glass eliminates corneal astigmatism. The field of vision of the person with myopia has been restricted, but through contact glasses he now has an increased visual field. Heine considered it important that contact glasses be put on children with low degrees of myopia to prevent progression of the defect.

As the contact glass adheres closely to the eye and participates in all its movements, it possesses various advantages over spectacles. Image-forming defects arising from the combination of the fixed lens with the moving system of the eye, such as astigmatism of oblique pencils of rays, alternation of perspective and, in a person with unequal sight in the two eyes, a difference in magnitude of the turning angle of the individual eyes, cannot occur. Rugg-Gunn has brought out some good points to show that for visual efficiency the contact glass has an advantage over the ordinary lens. Visual efficiency depends on monocular appreciation of perspective, binocular fusion and a wide field of vision. In correction with contact glasses the angle in the object space is identical with that in the image space. It is not always easy in anisometropia to bring the retinal images of the two eyes to a uniform size. With contact glasses, the retinal image is increased in the case of the myopic eye and decreased in the hypermetropic one, giving a similarity in the size and permitting of binocular fusion.

OPTICAL THEORY

In contrasting correction of ametropia with contact glasses with correction with ordinary spectacles, numerous optical problems arise. Among these phenomena, the size of the retinal image is a factor of great importance. Another factor of importance is that of displacement by means of optical correction of the cardinal points of the eye. Application of the science of optics proves that correction of the several varieties of ametropia with contact glasses involves a minimum displacement of the cardinal points, i. e., a minimum departure from the optical conditions of the normal eye.

SIZE OF RETINAL IMAGE

Ames, Gliddon and Ogle [1] called attention to the fact that, excluding pathologic conditions, one usually considers but two factors in determining whether the eyes of a person are functioning properly: first, the refraction, and second, the muscle balance. If there are emmetropia

1. Ames, A., Jr.; Gliddon, H. G., and Ogle, K. N.: Size and Shape of Ocular Images: I. Methods of Determination and Physiologic Significance. Arch. Ophth. **7**:576-597 (April) 1932.

and orthophoria for both distance and near vision, it is generally assumed that there is nothing the matter with the eyes as far as their functioning is concerned. There is a third factor, however, which appears to be of importance. This is the relative size and shape of the ocular images in the two eyes. The importance of this factor becomes evident when one considers the use of the eyes in binocular vision and the part played by the brain when the ocular images of the two eyes are combined. These authors go on to state that in order that the two images of the eyes be superimposed, three conditions are necessary: 1. The eyes must be looking in the same direction. 2. The images must be in focus; i. e., the refraction of both eyes must be correct. 3. The images must be the same size and shape. It can readily be demonstrated that any abnormal difference between the size or the shape of the ocular images in a horizontal direction deranges the apparent position of objects in the visual field. The simplest form of this derangement is an apparent horizontal rotation about a vertical axis at the point of fixation. The angular rotation in degrees caused by various percentages of disparity in size has been computed and tabulated. The perception of depth is a phenomenon that takes place in the brain, from which it follows that differences in the size and the shape of ocular images manifest themselves not in the eyes themselves but in the brain, where the images of both eyes are combined. Differences of size of the ocular images of 0.25 per cent can be distinguished, and differences of from 0.5 to 1 per cent should have about the same physiologic significance as an abnormal refractive condition of $\pm$ 0.25 to $\pm$ 0.5 D. Ames and his associates [1] made the generally known observation that associated with an abnormal difference in the size and shape of the ocular images there is a derangement of binocular vision. They mentioned a set of graded lenses having no effect on vergence of light but changing the size of the image from 0.25 to 5 per cent. Carleton and Madigan [2] emphasized the importance of prescribing lenses that correct not only errors of refraction but differences in the size of the image. Ames and Ogle [3] stated:

> It would seem reasonable to assume that those factors determining the adaptation to size differences between the eyes are very similar to those which determine the adaptation to refractive differences. The threshold values for the least perceptible differences in the refractive condition or for the monocular capacity to differentiate distance are near $\pm$ 0.12 diopters. Two or three times that amount is generally sufficient to cause ocular trouble. By analogy, two or three times the threshold value for size discrimination, that is, 0.50 per cent, should have the same effect.

2. Carleton, E. H., and Madigan, L. F.: Size and Shape of Ocular Images: II. Clinical Significance, Arch. Ophth. **7**:720-738 (May) 1932.

3. Ames, A., Jr., and Ogle, K. N.: Size and Shape of Ocular Images: III. Visual Sensitivity to Differences in the Relative Size of the Ocular Images of the Two Eyes, Arch. Ophth. **7**:904-924 (June) 1932.

The apparent size of an object is measured by the visual angle which it subtends at the eye. Variations in the size of this angle and consequently of the retinal image give rise, among other effects, to certain disturbances of monocular appreciation of perspective which are of practical importance. A person with myopia subtends a smaller angle and one with hypermetropia a larger angle than normal.

In correction with contact glasses, a magnification of the retinal image is a result of two factors: (*a*) that of the afocal contact glass and (*b*) that of the fluid meniscus in conjunction with axial length.

By the science of optics, one is able to confirm the clinical fact that in cases of monocular aphakia binocular fusion becomes possible when the other eye has useful vision. The difference in the size of the retinal images of the two eyes in aphakic anisometropia is not, however, the principal cause of diplopia. When the power of fusion is normal, the eye can achieve fusion with difference of at least 10 to 20 per cent. The chief difficulty arises from displacement of the image by the prismatic effect of spectacle lenses of high positive value. The eye can achieve fusion with differences in size, but one eye cannot deviate, say 10 degrees, and the other 30 degrees. With contact glasses, the necessity to do so does not exist, so that diplopia cannot occur either from incongruity of size of the image or from disturbance of the turning angle (*Blickwendewinkel*). With the higher degrees of myopia and, to a lesser extent, with the higher degrees of aphakia, a wide range of contact glasses is available as a basis on which a full correction can be prescribed by appropriate refractive corrections. This is a great advantage.

REPORT OF CASES

The difficulties usually encountered in the refraction of aphakic eyes, as described in the first paragraph, were impressed on me by the following cases:

Case 1.—M. S., a man aged 72, who consulted me, had been operated on in Vienna the previous year for monocular cataract and had traveled the world over seeking some one to prescribe a correction for his aphakic eye, even though his other eye was perfect. It was decided to attempt refraction of the aphakic eye (the left one) with contact glasses, and to the delight of all concerned, binocular single vision was restored. Vision in the right eye was 20/20; that in the left eye was 20/20 with correction.

Since the operative results in the left eye were so good, the patient was anxious to have the refractive error corrected so that he could use the eye in conjunction with the other eye, in which the vision was as good.

For the right eye a + 2.75 sph. ⊃ + .50 cyl., axis 175 gave vision of 20/20.

For the left eye a contact glass with a corneal curvature of 6 mm. and a + 1.00 sph. gave vision of 20/20. A + 2.25 cyl. was added for reading Jaeger's test type 1 and a 5 D. prism base in for exophoria.

Stereoscopic tests showed binocular single vision with fusion. A communication from the patient two months later stated that he wears his contact glasses for his eight hour business period.

The scientific explanation for this phenomenon was never clear until the principle of aniseikonia was promulgated. Hughes[4] stated that there is no question as to the enlargement of the image produced when the vision of an aphakic eye has been corrected by a lens of high plus strength. If the eyes were approximately emmetropic before extraction of the cataract, the difference in the size of the images of the two eyes amounts to nearly 30 per cent. This amount of aniseikonia can be corrected, but it is not practicable.

The value of correcting vision in monocular aphakia so as to produce binocular single vision is apparent. Traumatic cataract is common, and for persons with such cataracts to have the fusion faculty of the two eyes restored is a distinct advantage. The economic value in cases of compensation is apparent.

Correction with contact lenses in cases of monocular aphakia in all except the aged, to whom a blind eye is of no practical disadvantage, is recommended.

CASE 2.—J. A., aged 13 years, was operated on for a traumatic cataract six months previous to refraction with contact glasses. For the right eye a contact glass with a corneal curvature of 6 mm. gave vision of 20/20. For the left eye a —0.50 sph. ⊃ —0.50 cyl., axis 170 gave vision of 20/20.

Stereoscopic tests showed binocular single vision.

CASE 3.—P. C., a man aged 31, had a posterior polar and lamellar cataract in the left eye which had developed eleven years previously from an unknown cause.

Vision was limited to counting of fingers at 2 feet (61 cm.), while the vision of the right eye with correction was 20/15. The patient was desirous of being operated on because of his dislike for the cut in the field of vision and the blurred vision, which were handicaps in his business and sports. Other ophthalmologists had deferred operation, with the explanation that it would be impossible to have binocular vision. It was decided to extract the cataract and correct the vision in that eye with a contact glass. A simple intracapsular extraction was performed, followed by peripheral iridectomy. A contact glass with a corneal curve of 8.5 mm. and a + 13.000 cyl. ground on gave vision of 20/15. For the other eye a + 2.00 cyl., axis 90 gave vision of 20/15. An additional + 2.50 cyl. for this eye produced a reading vision for Jaeger's test type 1. Stereoscopic tests showed binocular single vision. The wearing time of the contact glass is sixteen hours daily. Iseikonic tests showed the image in the right eye to be 4 per cent smaller than that in the left eye, both vertically and horizontally. This difference is within the limits of fusion.

SUMMARY

A brief history as well as the chief uses for contact glasses are given.

The theoretic as well as the practical applications of the use of contact glasses are discussed.

4. Hughes, W. L.: Personal communication to the author.

It is shown how full correction may frequently be obtained by contact glasses in cases of monocular aphakia and how these glasses correct the disparity between the size and the focus of the images in the two eyes sufficiently to produce normal or nearly normal vision, including fusion and binocular vision, and to prevent diplopia—a result unattainable by correction with spectacles.

Three case reports are given illustrating the findings of the previous paragraph.

The use of contact lenses in correcting vision in cases of monocular aphakia adds one more important use for these aids to the correction of visual defects.

DISCUSSION

DR. CONRAD BERENS: I should like to know just what was the aptitude of fusion of the patient presented. Dr. Town referred to the fact that he has been able to obtain binocular single vision in some of these patients. I have found that by increasing the aptitude of fusion some patients were able to achieve fusion for distance. It is difficult for them to achieve fusion except with their eyes in the primary position. When they move the eyes up and down, the vertical prismatic displacement makes it difficult to maintain fusion. When I saw Dr. Town's patient the man was tired, but he did not seem to be able to achieve fusion at the near point.

DR. A. E. TOWN: The man was thought to be able to fuse images up to about 10 inches (25 cm.) from the eye—from infinity to 10 inches. With a + 3 diopter lens over his contact glass he was able to read with binocular single vision. He is not bothered by diplopia.

CREATINE AND CREATININE OF THE OCULAR TISSUES

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AND
F. WARREN TAUBER
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Creatine and its anhydride, creatinine, are important substances which take part in the metabolism of tissue.[1] In vertebrates creatine occurs in the tissue as creatine phosphate and free creatine, but in invertebrates the creatine is replaced by arginine. The question whether or not the precursor of creatine is arginine in vertebrates is unsettled, although creatine is related to arginine metabolically and structurally. Creatine is converted into creatinine by the tissue. The quantity of creatine in the tissue is relatively large as compared to that of creatinine. There seems to be no doubt that the formation and cleavage of creatine phosphate is a cyclic process which is concerned with the carbohydrate metabolism of active tissue. Apparently it is primarily associated with the coupling of dextrose to phosphate in the formation of dextrose phosphate as the first step in the glycolysis of dextrose. The dextrose phosphate is changed into a more active molecule, and after coupling to more phosphate the molecule is split into two parts. The end product is lactic acid. The carbohydrate metabolism is abnormal, and there is a loss of tissue creatine in certain pathologic conditions leading to atrophy of tissues, such as in myopathies, high fevers, starvation, severe diabetes and hyperthyroidism.

The interest in the creatine of the eye has been secondary to that of other substances. Krause and Yudkin[2] reported that 1.7 mg. of creatine per hundred cubic centimeters of aqueous occurred in the aqueous humor of the dog. Cohen, Killian and Metzger[3] found 1.6 mg.

From the Division of Ophthalmology, Department of Surgery, University of Chicago.

1. Hunter, A.: Creatine and Creatinine, London, Longmans, Green & Company, 1928. Thomas, K.: The Metabolism of Creatine and Creatinine, in Luck, J. M., and Noller, C. R.: Annual Review of Biochemistry, Stanford University, Calif., Stanford University Press, 1938, vol. 7, p. 211.

2. Krause, A. C., and Yudkin, A. M.: The Chemical Composition of the Normal Aqueous Humor of the Dog, J. Biol. Chem. **88**:471, 1930.

3. Cohen, M.; Killian, J. A., and Metzger, N.: Studies in the Chemistry of the Humors of Animal Eyes, and a Comparison of Their Composition with That

(Footnote continued on next page)

of creatine and 1.1 mg of creatinine per hundred cubic centimeters in the vitreous humor of the ox, and Duke-Elder,[4] 1.0 mg. of creatine per hundred cubic centimeters in the vitreous humor of the horse. Stilo[5] noted that small amounts of both substances occurred in the retina. Rizzo[6] estimated that the lens contained 1 mg. of creatinine and about 15 mg. of creatine per hundred cubic centimeters.

It is evident that creatine is involved in the normal and pathologic metabolism of ocular tissue. The supposition is that it behaves in this tissue in the same manner as it does in other tissue, although practically nothing is known specifically about creatine in ocular tissue. It is probable that creatine metabolism may be related to cataract in dystrophia myotonica, to atrophy of the ocular muscles in myasthenia gravis and to other ocular diseases. Because the knowledge of ocular creatine is so meager, this investigation was made.

EXPERIMENTAL PROCEDURE

The methods which are commonly used for the estimation of creatine and creatinine in tissues are based on relatively unspecific reactions which yield colored substances. In most tissue extracts the amount of color arising from the reaction exceeds that which corresponds to the quantity of creatinine. The method which keeps the secondary color formation to a minimum and which gives reproducible results is the one to be used. The method of Rose, Helmer and Chanutin[7] gave good results with muscle but not with most of the ocular tissues. The procedure of Benedict and Behre[8] and of Langley and Evans,[9] using 3,5-dinitrobenzoic acid, was found to be applicable to these tissues.

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of Blood and Spinal Fluid, in Contributions to Ophthalmic Science, Dedicated to Dr. Edward Jackson, in Honor of His Seventieth Birthday, Menasha, Wis., George Banta Publishing Company, 1926, pp. 216-228.

4. Duke-Elder, W. S.: The Nature of the Vitreous Body, London, Geo. Pulman & Sons, Ltd., 1930.

5. Stilo, A.: Biochemistry of the Retina: I. Soluble Phosphorus of Bovine Retina, Boll. Soc. ital. di biol. sper. **12**:806, 1937; abstracted, Chem. Abstr. **32**: 2194, 1938.

6. Rizzo, A.: Biochemistry of the Crystalline Lens: I. Presence of Labile Phosphorus and Behavior of Other Complex Phosphates, Boll. Soc. ital. di biol. sper. **12**:810, 1937; abstracted, Chem. Abstr. **32**:2194, 1938.

7. Rose, W. C.; Helmer, O. M., and Chanutin, A.: A Modified Method for the Estimation of Total Creatinine in Small Amounts of Tissues, J. Biol. Chem. **75**:543, 1927.

8. Benedict, S. R., and Behre, J. A.: Some Applications of a New Color Reaction for Creatinine, J. Biol. Chem. **114**:515, 1936.

9. Langley, W. D., and Evans, M.: The Determination of Creatinine with Sodium 3,5-Dinitrobenzoate, J. Biol. Chem. **115**:333, 1936.

A brief description of the procedure which was used is given:[10]

Samples of 10 to 20 Gm. of cold fresh dissected tissue were finely minced and then mixed with 5 to 10 volumes of water. The proteins were precipitated with tungstic acid in the usual manner. After the preparation was stirred for twenty minutes, the proteins were filtered off. To an aliquot of the filtrate, 3 cc. of concentrated hydrochloric acid was added. The solution was placed in a steam autoclave for forty-five minutes at 15 to 20 pounds (6.8 to 9 Kg.) of pressure. After the solution cooled, it was neutralized with sodium hydroxide, then diluted and filtered. Sufficient water was added to 2 to 5 cc. of the filtrate to make a volume of 10 cc. Three cubic centimeters of a saturated solution of sodium dinitrobenzoate and 0.5 cc. of a 10 per cent solution of sodium hydroxide were added. After ten minutes the solution was measured in a Zeiss Pulfrich photometer with a S50 filter and 1 cm. cups. The data were corrected by reference to a curve obtained from calibration of the instrument with a standard solution of creatinine containing 0.7 to 2 mg. per hundred cubic centimeters. Determinations were made on 30 samples of retina, 4 of lens and 8 of each of the other tissues.

The enzymatic method for analysis of creatine, which was performed by Dr. B. Miller in the medical department, gave an average of 59 mg. of creatine per hundred grams of tissue in three samples of retina and 12.3 mg. in three samples of lens. These results were in agreement with those obtained by the Benedict method.

When it was possible, the isolation and identification of creatinine potassium picrate were done according to the technic of Gaebler and Keltch.[11] Creatinine zinc chloride was prepared from the picrate and identified. For isolation of salts, samples of ocular tissue weighing from 200 to 1,000 Gm. were used. Creatinine zinc chloride made from lens creatine was found to have 26.74 per cent carbon, 3.72 per cent hydrogen and 22.86 and 22.95 per cent nitrogen as compared with 26.5 per cent carbon, 3.86 per cent hydrogen and 23.13 per cent nitrogen calculated for the salt.

COMMENT

The total amount of creatine and creatinine as chromogenic substance in fresh bovine ocular tissues is given in table 1 and the amount of creatinine in table 2. By subtracting the amount of creatinine from the total creatinine, the quantity of creatine may be found.

Obviously only a part of the color arises from creatinine. The presence of other secondary chromogenic substances vary with the kind of tissue. Although 70 to 90 per cent of the creatinine added to tissue may be recovered, the estimation by isolation of the salt in fresh tissue is worthless quantitatively because an excessive and irregular loss of creatinine occurs but is of value qualitatively, for it proves that the substance is actually present.

10. Benedict and Behre.[8] Langley and Evans.[9]

11. Gaebler, O. H., and Keltch, A. K.: On the Nature of Blood Creatinine, J. Biol. Chem. **76**:337, 1928.

The creatine content gives a clue to the metabolic activity of tissue. Sclera, corneal stroma and vitreous humor which are relatively inert have little creatine. The other ocular tissues, such as corneal epithelium and uveal tissue, have a much higher content and are more active. A discussion of the creatine metabolism in the lens is given in another paper.[12] There is reason to believe that the creatine and creatinine metabolism of ocular tissue is similar to that of other tissue and is modified according to its function.

TABLE 1.—*Chromogenic Substance Expressed as Total Creatinine (Creatine Plus Creatinine) in Fresh Bovine Ocular Tissues*

	Mg. per 100 Gm. of Fresh Tissue			
	Minimum	Maximum	Average	Chromogenic Substance Isolated as Creatinine
Cornea	26.6	34.5	30.8	14.0
Epithelium	59.3	84.4	70.9	....
Stroma	12.6	19.3	16.6	....
Sclera	8.6	14.8	9.3	....
Iris	45.0	69.7	47.5	16.1
Choroid	39.6	58.4	44.3	....
Lens				
Cortex	15.3	16.8	16.1	....
Nucleus	13.4	14.5	13.9	....
Calf	14.2	19.7	16.6	....
Steer	10.4	16.2	13.3	1.9
Vitreous humor	3.0	6.4	5.1	....
Retina	48.4	80.7	69.5	8.7
Optic nerve	61.6	96.0	69.5	15.7

TABLE 2.—*Creatinine in Fresh Bovine Ocular Tissues*

	Free Creatinine, Mg. per 100 Gm. of Tissue		
Tissue	Minimum	Maximum	Average
Conjunctiva	1.1	1.9	1.5
Cornea			
Whole	0.95	1.2	1.1
Epithelium	11.3	14.6	12.8
Stroma	1.0	1.2	1.1
Sclera	0.85	1.07	0 9
Iris	1.1	1.3	1.2
Choroid	2.1	2.5	2.3
Lens	0.86	1.2	1.0
Vitreous humor	0.85	1.1	0.96
Retina	1.3	1.5	1.4
Optic nerve	0.49	0.63	0.6

SUMMARY

Colorimetric determinations of creatinine and total apparent creatinine (creatine plus creatinine) of the ocular tissues are reported. Creatinine of lens, iris, retina, optic nerve and cornea was isolated as the potassium picrate. The significance of creatine in the ocular tissues with special reference to the metabolism of lens is discussed.

12. Krause, A. C.: Chemistry of the Lens: VIII. Lenticular Metabolism. Arch. Ophth. **17**:468 (March) 1937.

Clinical Notes

CIRCUMCORNEAL TELANGIECTASIS

Report of a Case

MARVIN J. BLAESS, M.D., DETROIT

A rare anomaly which produces a "red eye" and has not yet been recorded in the ophthalmic literature is presented in order to record the occurrence of this condition as a pathologic entity and to bring forth its consideration in differential diagnosis.

REPORT OF CASE

A dental surgeon, aged 54, white, complained of redness of the right eye and a gradual diminution of visual acuity in this eye for three years. There had always been some redness in the eye since birth. It would disappear after the instillation of a few drops of epinephrine hydrochloride. There was no history of injury, sensitization, dietary restrictions or familial ocular disease. The patient was quite impressed by the fact that for many years in his professional work he had used an intense light from a lamp that was placed directly in front of the right eye and caused a continuous glare therein. The vision in his left eye remained as good as ever. He had received treatment for "iritis" on a number of occasions in the preceding years.

On the skin of the right side of the head and face, extending from the region of the right temple diagonally down across the right upper eyelid to the bridge of the nose, were scattered irregular-shaped, mottled, deep purplish red patches of various sizes (a naevus flammeus). One of these patches occurred at the middle of the right upper lid and another at the right outer canthus. There was a divergence of the right eye of 10 degrees, but there was good motility in all directions. The convergence of the left eye was normal, but the right eye did not converge at all. No pain or tenderness was elicited on digital pressure, and tactile tension was normally soft. With the Gradle-Schiötz tonometer the tension in the right eye was 17 mm. and in the left 28 mm.; the pupils measured 2.5 mm. in diameter. The left eye appeared normal. The right eye appeared red, and with the Beebe loupe it showed an area of peculiar punctate circumcorneal injection, wider below the cornea than above and tapering from the limbus on each side, where it measured 10 mm. in width to about 1 mm. at each canthus. The appearance of the eye is shown in the accompanying illustration. The reddened area corresponded roughly to the palpebral fissure. The conjunctiva in this area was loose, forming several small redundant folds which extended between the margins of the upper and lower lids when the lids were closed. The remaining bulbar and the palpebral conjunctiva were normally pale.

The right lens was entirely opaque in the small central pupillary area and had a grayish white appearance. The ophthalmoscope did not reveal even the faintest red fundus reflex in the right eye. Through the small pupil the left lens showed a definite clouding in the outer layers of the lower half, but the vitreous was clear, and through the upper half of the lens the fundus and the disk appeared normal.

With mydriatics the pupil of each eye dilated evenly to 7 mm. The opacity of the left lens involved the lower half of the lens, and the right lens was entirely opaque and had a smaller, round, brownish, eccentric intralenticular area below. No red fundus reflex was observed in the right eye. The left fundus was normal, and no abnormal vascular, atrophic, pigmentary or inflammatory changes were discernible.

With the narrow beam of the slit lamp the conjunctival prism in the limbic region of the right eye was clearly visible and broader than is usual. The conjunctival epithelium was thinner than normal, and the subconjunctival portion of the beam was broadened by the chemosis of the subconjunctival tissues. Here could be seen a broad and unusually extensive superficial marginal plexus in the right episcleral tissue formed by a profusely anastomosing arterial network of vessels. The network was arranged at the limbus in a series of closely knit terminal arcades which overlaid the deeper venous plexus formed from the final marginal vessel loops of the arterial net. The vessels of this dense superficial marginal plexus made a striking picture as they appeared irregularly dilated or

Appearance of the patient's right eye.

pouched at intervals along their courses. The vascular anomaly itself was varied, with the defects ranging from small ectasia to large blind diverticulae of many different sizes and shapes and all brilliant red. In some places the ectasia involved only a part of the wall of the vessel, producing a pouch. In other places the entire vessel would be markedly dilated, appearing as a miniature aneurysm. At the limbus the general disposition of the arcade arrangement of the superficial marginal plexus was maintained, with the anastomosing vessels showing variations of the same ectatic anomaly. At a distance of 0.5 to 2 mm. from the limbus, however, the regular arcade arrangement rapidly passed into a loose and tangled nondescript mass of vessels (angioma telangiectoides) in the episcleral tissue.

The vessels ranged in size from small thin-walled tubes, in which red cells could be seen flowing in single file, to large thin-walled spaces 20 to 30 red cell diameters in caliber, in which the cells moved en masse. Hence in the limbic arcade and terminal vessels the condition could be specifically referred to as multiple hemangiectasis of the capillaries and arterioles, but in the episcleral tissues beyond the limbus the anomaly was clearly that of an angioma telangiectoides. In the blind pouches and diverticulae there was the punctate circumcorneal injection seen macroscopically. The vessels of the underlying venous plexus were of a deep purplish red and definitely larger in caliber and more richly anastomotic

than normal, but they were of uniform diameter and did not show the irregular intermittent ectatic character of the vessels of the superficial arterial plexus.

The tissue interspaces between the plexal vessels were wide, and the depth of the subconjunctival space above and below the arterial portion of the superficial marginal plexus was definitely increased. However, when the eye was examined with a red-free light there was no increase in cellular elements of either vascular or tissue origin and no extravasated red blood cells. These widened tissue interspaces were optically empty, being occupied by a clear, colorless fluid. No lymph vessels could be made out, even with a yellow filter.

After three instillations of a solution of epinephrine hydrochloride (1:1,000) at intervals of five minutes, all redness in the right eye would disappear, leaving a glistening white, slightly chemotic conjunctiva. With the slit lamp only an occasional bright red arterial vessel could be made out, but the more purplish red, deeper venous plexus was still clearly visible.

Vision in the right eye was limited to perception of hand movements at 18 inches (46 cm.); vision in the left eye was 20/100 — 1. By manifest refraction the vision in the right eye could not be improved, but with a —2.00 sphere the visual acuity and fields of the left eye were normal.

Projection of light was quick and accurate in all quadrants at 4 feet (122 cm.) and at 20 feet (6 meters) with the right eye. Color vision in the left eye was normal, but an intense red was the only color recognizable with the right eye.

Complete physical examination and extensive laboratory tests revealed no pertinent abnormalities. Microscopic examination of stained conjunctival smears failed to reveal any unusual pathogenic organisms. The patient was given 0.01 Gm. of ascorbic acid and 3 Gm. of calcium gluconate daily as possible prophylaxis against excessive hemorrhage previous to the extraction of the right lens.

I removed the cataract intact in its capsule, using a modified combined Barraquer suction technic. Operative hemorrhage was controlled by the liberal use of a solution of epinephrine hydrochloride.

I have examined this patient at intervals for two years since the operation. With a + 12.00 sphere the visual acuity in the right eye is 20/20. The circumcorneal telangiectasis in this eye has remained unchanged, except for the minor alteration due to the corneal section, and the eye still appears red. The choroidal blood vessels were not involved in the vascular anomaly.

A FEATHERWEIGHT SLIT LAMP

EMANUEL KRIMSKY, M.D., BROOKLYN

In the August 1932 issue of the ARCHIVES I described an illuminated loupe.[1] In this article I stated:

One of the difficulties confronting the ophthalmologist in the use of the monocular loupe is in obtaining a sufficient amount of condensed light at a suitable focal distance. Besides the fact that one hand is occupied in holding the condensing lens, the source of the illumination may not always be close or conveniently at hand. In order to overcome these shortcomings it occurred to me that if the light

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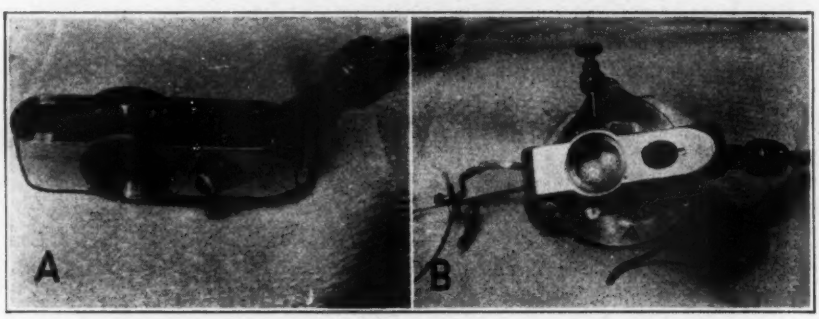
Presented before the New York Society for Clinical Ophthalmology, Oct. 3, 1938.

1. Krimsky, E.: A Modified Monocular Loupe, Arch. Ophth. 8:272-274 (Aug.) 1932.

source and condenser could be combined as a unit and this in turn attached or fastened to the loupe, a satisfactory and reliable source of illumination might be effected to assist the operator in his observations.

I also described how this crude model could be used as a hand slit lamp.

In designing a slit lamp for hand use, the problem was to provide me with a so-called featherweight instrument that could be carried in the hand and would not interfere with the free use of the ophthalmoscope. Hence, besides being a light instrument, it had to be an independent unit, as the use of a common battery handle for both instruments might prove time consuming and expose them to breakage through frequent interchange. I have therefore found it practical to respect the intactness of the ophthalmoscope with its battery handle and to supply the slit lamp with a separate medium-sized battery handle (carried in the coat pocket), using no. 935 batteries connected to the slit lamp by means of a fine silk-woven wire. Such an arrangement permits the slit lamp to be slipped back into the coat pocket, adds to its lightness by not being joined directly to the handle, obviates frequent



A, the slit lamp proper. *B*, the slit lamp with attachment.

disturbance of the ophthalmoscope, is time saving and permits the lamp to be held between two fingers instead of by two hands. Furthermore, as will be shown later, by the attachment of a spring clamp to the wearer's eyeglass both hands may be entirely free to permit use of instruments in such conditions as the removal of foreign bodies or when the other uses are demanded of the operator's hands.

This new slit lamp, like its forerunner, has been designed to include the following features: (1) centralization and proper angulation of light at an angle of 45 degrees, (2) firmness, (3) lightness, (4) compactness and simplicity of operation and (5) proper quality and quantity of illumination. In addition, the newer features are: (1) an automatic device to change the beam progressively from a vertical slit to a transverse slit and finally to a homogeneous circle of light, (2) an attachment to permit the instrument to be worn over the eyeglass and (3) a slit lamp unit rather than a modified loupe.

The features of this lamp in more detail follow:

1. The slit lamp proper consists of a lens housing made of duralumin, at one end of which is a lens mount containing an improved Zeiss 10 × double aplanatic lens, and at the other, a channel which makes a 45 degree angle with the focal axis which holds the lamp unit.

2. The lamp unit consists of a light carrier, a straight filament bulb and a hemispherical condenser. This unit has been made to provide automatically and progressively a vertical slit, a horizontal slit and a homogeneous circle of light by the simple turning of the base of the unit which has had incorporated in it a helical mount, which operates in such a way as to slide the bulb backward or forward without displacing the condenser. By such manipulation, the approximation of bulb and condenser produces a circle of light, and separation leads to a slit light by bringing the filament in the focal plane of the condenser.

3. The slit lamp proper, including the light carrier, weighs but 29 Gm. (less than 1 ounce). The addition of the spring clamp brings its weight up to 37 Gm.

4. The spring clamp consists of a pair of clamps so constructed that the eyeglass can be slipped between them and held in place. This clamp has at its upper part a horizontal rod which extends back to meet a vertically projecting rod that joins the slit lamp proper. When not in use, this clamp can be easily freed by means of a fastening screw.

5. A fixed angulation of the light carrier at 45 degrees to the axis of the lens was found most desirable for practical requirements.

6. While a 10 × Zeiss lens was used in this instrument, it would be preferable in cases in which an attachable clamp is worn frequently to have a weaker glass (6 ×) which increases the focal length or distance from the eyes sufficiently to allow freer use of the hands.

This entire unit can be easily disassembled. Moreover, by removal of lamp unit from the lens housing proper, the operator can, by manipulating the condenser, adjust the slit to satisfy his particular requirements.

GONORRHEAL OPHTHALMIA CURED AFTER ONE WEEK'S TREATMENT WITH SULFANILAMIDE

S. HANFORD McKEE, M.D., MONTREAL, CANADA

Sulfanilamide was first recommended to the medical profession as a therapeutic agent in infections due to Streptococcus haemolyticus of the beta type. Its use has, however, widened materially from that concept. Reports from hospitals and in the general literature indicate how extensive its use has become. They warn too that such therapy is not without danger and that caution and attention to the blood picture during the administration of the drug are most necessary. Unquestionably it is a valuable therapeutic agent, yet much remains to be learned regarding its use in ophthalmic infections.

Reports in this connection are desired, that the value of the drug may be properly estimated, and for this reason the following report of a case of gonorrheal ophthalmia is submitted. It is fully realized that what has taken place in one case may give little real indication, but taken with the results in other cases it may help to establish either the value or the uselessness of the remedy in this disease.

From the Departments of Ophthalmology and Pathology, Montreal General Hospital.

Fernandez and Fernandez [1] recently reported on sulfanilamide in the treatment of gonorrheal ophthalmia. The following statements are taken from their conclusions:

All patients who received sulfanilamide recovered in a spectacular manner and in a shorter period of time than that required by other accepted forms of treatment.

The results obtained in this series of cases warrant the judicious use of sulfanilamide in all cases of gonorrheal ophthalmia in adults, whenever there is no serious contraindication.

It is highly desirable that this method of treatment be tested by our colleagues to corroborate our findings.

Smaller doses and special precautions must be used for patients with renal insufficiency, because the excretion of sulfanilamide is slow, and in such patients would tend towards accumulation of the drug in the blood.

The report of my case follows:

REPORT OF A CASE

R. D., a man aged 23, was seen in the late afternoon of Dec. 29, 1938, complaining of discomfort in his right eye, which was found to be intensely inflamed. The lids were swollen, the bulbar conjunctiva was chemotic, and there was a profuse purulent discharge. The preauricular gland was swollen and tender. A smear made from the discharge revealed innumerable gram-negative, coffee-bean-shaped, intracellular diplococci. The patient stated that his eye had been inflamed for a number of days but that in the last two it had become intensely red and swollen, with a copious discharge of pus. At the time of examination it presented the typical appearance of severe gonorrheal ophthalmia. It was found that he had an attack of urethritis as well, which had been present for a number of weeks.

He was sent to the isolation ward of the ophthalmic service, where the usual treatment of frequent irrigations with a warm solution of boric acid followed by the instillation of drops of a 25 per cent solution of mild protein silver was begun; a Buller shield was supplied to the unaffected eye. To this local treatment was added the internal administration of 15 grains (0.972 gm.) of sulfanilamide with 5 grains (0.324 Gm.) of sodium bicarbonate every six hours.

The patient's progress was remarkable. On the third day the discharge was noticeably less, and on the fourth day the infection was improving in a most satisfactory manner. A culture which had been taken on hemoglobin agar from the conjunctival discharge on the day of the patient's admission was reported by the pathologic department to be positive for gonococci. On the fifth day of treatment the discharge had almost disappeared, and a prolonged search for gonococci in an epithelial cell smear gave negative results. Two days later the patient was discharged well.

Previously, infections of this kind entailed the employment of two graduate nurses for five to six weeks. Some years ago I [2] reported the cultivation of the gonococcus on the thirty-fifth day of hospital treatment in a similar case of ophthalmia in an adult. In the case reported here prolonged search failed to reveal gonococci on the fifth day.

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1. Fernandez, L. J., and Fernandez, R. F.: Sulfanilamide in Gonorrheal Ophthalmia: Preliminary Report, Am. J. Ophth. **21**:763 (July) 1938.

2. McKee, S. H.: Bacterial Phagocytosis by the Epithelial Cells of the Conjunctiva, Canad. M. A. J. **15**:703 (July) 1925.

Ophthalmologic Reviews

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ARRESTS IN EMBRYOLOGIC DEVELOPMENT AS FACTORS IN VISION

BRIEF REVIEW OF THE EMBRYOLOGY OF THE EYE WITH ASSOCIATED ANOMALIES OF ARRESTED DEVELOPMENT

DONALD J. LYLE, M.D.

CINCINNATI

The embryologic development of the human eye is a comprehensive subject of study. If one wanders into the more or less speculative fields of genetics to account for the numerous varieties of eyes, even though the differences remain within type, the subject and the interest broaden. It is impossible in this short review to do more than outline the essential embryologic development of the human eye. Such an outline is absolutely necessary, sketchy as it is, in order to discuss clearly, so far as the present knowledge of the subject will permit, the conditions caused by arrest in development during various periods in embryologic growth.

If intrauterine inflammation and birth injury are excluded, it is probably correct to state that most congenital defects are due to aberrations in development. These may consist of underdevelopment, overdevelopment or perverted development. Of these, underdevelopment or arrested development, appears most common. These arrests, though they should not show a departure from normal for that period, often result in secondary changes in the tissues arrested and in the surroundings. Frequently the reaction is a readjustment to the arrest by compensatory changes which resemble anomalies or abnormalities.

There are a number of factors which may affect and check the developing eye, or arrest it, in its normal growth. The cause may be germinal, in which case there is usually a hereditary transmission pattern. Occasional causes of arrest are infections, poisons, toxins, metabolic and endocrine disturbances in the mother acting on the developing eye through the placental circulation and mechanical injury or pressure which directly interferes with the normal growth of the embryonic eye.

It is not definitely known at present the part some of these factors play in causing arrest of development in the embryonic eye; for instance, shock to the mother which checks the proper blood supply to the eye.

Involvement of the mother's eyes due to injury and inflammation and the effect of these factors on the embryonic eye by the production of antibodies must also be considered. Would sympathetic ophthalmia in the mother involve the fetal eye? Would the properties developed as the cause or result of maternal uveitis from disease or absorption of lens cortex from trauma affect the developing eye?

There are frequently two phases in the life of the embryologic structures of the eye, especially the mesodermal tissues, a stage of development or growth and a period of recession or atrophy. This is most clearly demonstrated by the intraocular vascular system in which there is a complex and prolific development of the hyaloid system and vascular tunic of the lens and then almost complete disappearance of these vessels by birth. An arrest may occur during a developmental stage or during a recessive period, resulting in the appearance of an earlier developmental stage or a later period of atrophy.

The eye begins developing soon after fertilization. It is subject therefore, over a long period, to various influences. It is evident that the earlier the disturbance the more pronounced is the effect on the developing eye. Along with these earlier, and grosser, aberrations, the eye is only one of the many structures involved, so that frequently life is not possible (i. e., cyclops).

ARRESTS IN DEVELOPMENT OF ECTODERMAL STRUCTURES

The neural plate, the beginning of the central nervous system, changes to the neural groove (later the neural tube), in the forward extension of which the head fold (forebrain) forms. The eye-forming material is located in the anterior median part of the neural plate. As the neural plate widens and forms a groove, this median anlage divides, pits and separates to form the optic vesicles, which remain joined to the brain by the optic stalks.[1]

The earliest arrest in the development of the eye takes place when there is total failure of the optic vesicle to push out from the forebrain. The result is the absences of an eye, which is termed anophthalmia (fig. 1 *A*). Examination in such a case reveals that there is no neural ectodermal tissue in the orbit. Some authors state that the bony orbit is of smaller size than normal. I have measured the orbits of two anophthalmic eyes and a number of orbits of microphthalmic eyes and have found them to be of normal dimensions. The orbit, eyelids, lacrimal

1. There are two interpretations of the inception of the optic vesicles from the forebrain. One group believes that there is one spot of eye-forming material situated in the median part of the anterior end of the neural plate. Another group believes that there are originally two (bilateral) spots of eye-forming material on both sides of the median region of the neural plate.

apparatus, conjunctival sac and extrinsic ocular muscles are all self determined and do not need the stimulus of the optic vesicle to incite growth. Therefore, the mesodermal tissues which go to form the parts of the orbit and globe are present and make a normal effort to carry out their development, the result being a small nubbin of mesodermal tissue in the place of an eye. The scaffolding, the optic vesicle, is absent (fig. 1 *B*).

The optic anlage may push out from the forebrain but may fail to separate into the paired vesicles, or it may be arrested at the beginning of its separation. (There are many grades of arrested separation.)

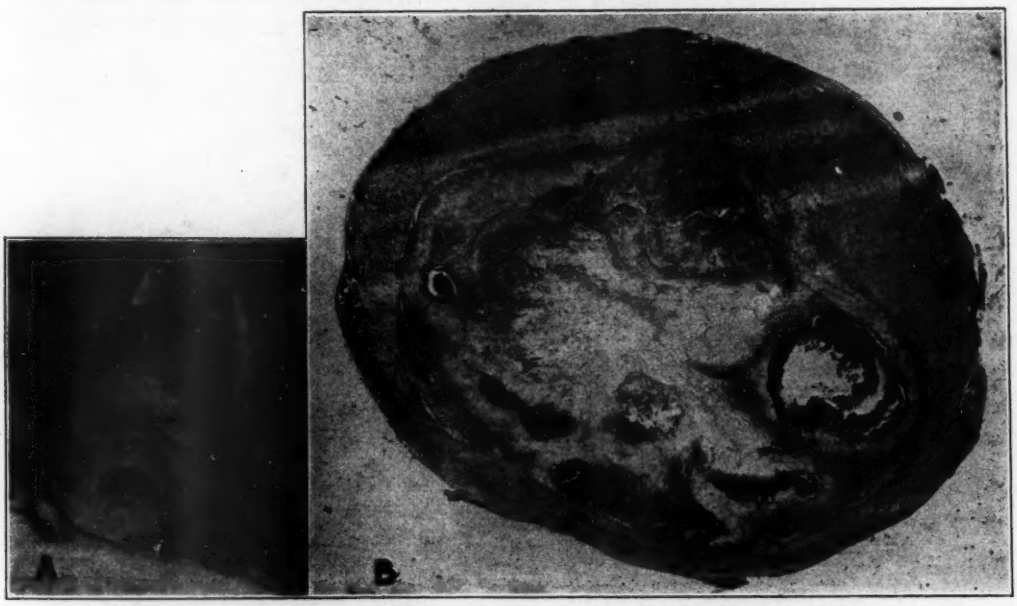


Fig. 1.—*A,* anophthalmia, due to arrest in the development of the neural ectodermal structures. The small nubbin (*B*) removed from the orbit showed essentially mesodermal tissue which had made an effort to carry out its part in forming an eye.

In this median position the vesicle or fused vesicles develop into the fused cyclopean eye (synophthalmia). This condition may be due to the lack of power on the part of the neural plate or the substrate to divide or separate normally, or if there are originally two centers there may be fusion because of defective development between them. The failure of separation of the eyes and orbits results in the pushing forward of the frontonasal process, which, hanging down from above, forms a proboscis. As in the specimen pictured in figure 2, many cyclops show failure in lateral or paired development in other structures. In

this cyclops there was arrest in closure of the neural tube and absence of cerebral hemispheres among other anomalies.

In the first few weeks of embryonic development the optic vesicle grows laterally from the primitive brain and approaches the surface ectoderm, where it incites the formation of the lens. Then the vesicle invaginates to form the optic cup. If the optic vesicle fails to invaginate, it may undergo cystic degeneration and dilatation, resulting in the congenital cystic eyeball. The anterior part of the cyst shows the retinal elements of the normally invaginated optic cup, and the posterior part contains pigment of the outer wall of the cup.

With normal development, the under side of the cupping optic vesicle and stalk (the narrowing connection between vesicle and brain) invaginates still farther into a groove which extends from the edge of the cup

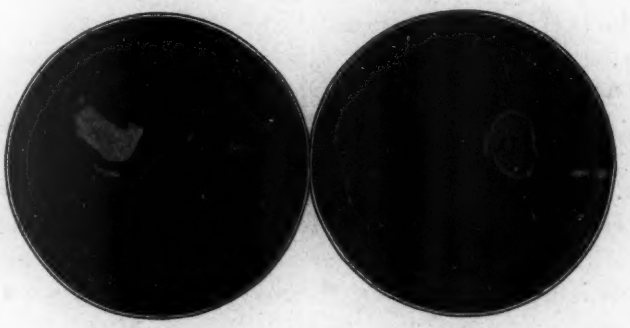
Fig. 2.—*A*, cyclopia (synophthalmia), due to arrest in the development of the midline structures, probably ectodermal and mesodermal. *B*, a section through the orbit showing fused globes with a single optic nerve composed of fibers from both retinas with a single sheath.

to the forebrain. This is called the fetal fissure (choroidal fissure). It begins to close at its center, and then closure proceeds forward and backward, reaching the papillary area at the beginning of the second month. At about this time the outer wall of the cup shows pigmentation.

If arrest in the normal development occurs before the fetal fissure has closed, two types of cysts, usually associated with microphthalmia, may develop. The first type is due to the growth of ectodermal tissue through the fissure. Sometimes more than one cyst is formed. The cyst is lined with nerve and pigment elements of the anterior and posterior parts of the wall of the optic vesicle and communicates with the cavity of the cup (vitreous cavity). The other type of cyst forms

between the inner nerve layer and the pigmented outer layer of the cup and does not connect with the space within the cup (fig. 3*A*). This cyst is a dilated part of the primary optic vesicle. Other cystic dilatations of the primary optic vesicle occur at the margin of the cup later, long after the fetal fissure has closed.

There is still another condition found in the area of the fissure in eyes arrested at this stage of development. In this case there is no herniation or extension sufficient to form a cyst, but there is a closing over of the patent fetal fissure, usually with some ectasia. This defect is called a coloboma and may be found involving the whole or only a small part of the fetal fissure. It may indirectly cause the typical coloboma of the iris and ciliary body which develop after closure of the normal fissure. It causes the formation of coloboma of the retina (in reality the choroid) and the optic nerve. Factors resulting in the formation of a typical coloboma are not thoroughly understood. Coloboma is



A B

Fig. 3.—*A*, microphthalmia with a cyst formed between the two layers of the optic cup. *B*, buphthalmia, resulting in deficient drainage from arrest in the development of the canal of Schlemm and arrest in absorption of mesoderm from the iridocorneal angle. This section also shows the fetal arrest in the iris and ciliary body.

probably more than a failure in the closure of the fetal fissure. A typical coloboma usually appears as follows: The floor is usually white, irregular, ectatic and lined with pigment. No choriocapillaries are found and but few choroidal vessels. Retinal vessels generally pass over the coloboma unchanged. The sclera is thin, and the lamina fusca with deep choroidal vessels is frequently present. There is no membrane of Bruch. The retina is atrophic. It has been shown that there is no true gap in the retina. Frequently there has been found a lapping of two layers of retina, which are seen as a mere membrane containing undeveloped or degenerated nerve elements but no pigment.

Some observers have concluded that the coloboma is probably due to an excessive eversion of the inner nerve layer of the optic cup so that it presents itself to the outside of the cup, folding the pigment layer

backward on itself and covering it on the outside with retinal tissue. It has been shown that the choriocapillaries of the choroid develop only from mesoderm which is in contact with pigment epithelium. As there is no pigment epithelium along the fissure or its margins, where it is covered with ectatic retina, the contact is not at hand to incite choroidal development over this area and no blood is supplied to the tissues, with the resultant arrest and degeneration of those tissues already formed.

As to the cause of arrest in closure of the fetal fissure, there is some division of thought, as proof is lacking. Most authors today believe that there is a delay in the closure of the fissure due to the presence of mesodermal tissue between its margins. In early stages vessels normally enter the fissure, as does the hyaloid artery. Some mesodermal stroma also enters. But the mesodermal tissue in the fissure, except the hyaloid artery which is walled off in the stalk, should offer no resistance to closure and should be pushed aside by the closing fissure. However, this delay of mesodermal tissue to get out of the way in time, thus resulting in aberrations, is found elsewhere in the developing eye, as shown by the formation of polar cataracts, a persistent pupillary membrane and a persistent hyaloid artery.

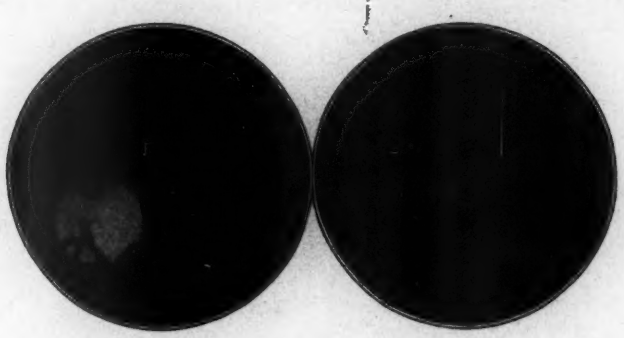
It may be that instead of mesodermal tissue filling the fetal fissure, preventing its closure, ectodermal tissue is the primary factor in causing colobomas. It is known that the inner layer of the optic cup develops much faster than the outer layer. If the development is greatly out of proportion, the inner layer must, of necessity, be thrown into ridges or folds. This is noted in many sections of embryonic eyes. If the fetal fissure is present at this time, as it usually is, there is nothing, save possibly invading mesoderm, to prevent the protrusion of the excessive inner layer into or through the fissure. If the amount protruding is slight, a coloboma is formed; if it is great, cysts are formed.

This overdevelopment of the inner layer of the retina in lesser amount may result in wrinkling, causing the light reflexes found in hypermetropia. If the disproportion or the development is still more exaggerated, it may cause folds and septums in the retina and may account for congenital detachment of the retina.

It might be well to mention macular dysplasia or coloboma. Examination of eyes with such a defect reveals the constant absence of the layer of choriocapillaries, though in certain types of dysplasia there seems to have been hyperplasia of this layer with pigmentation. The deep choroid and sclera may be affected, in which case ectasia frequently results. The deep retina is occasionally involved (fig. 4 *A*).

Such dysplasias are, of course, not in the area of the fetal fissure, so they cannot be typical colobomas. There are a number of theories concerning their inception, the chief ones being based on local ectodermal or mesodermal arrest or failure, and on hemorrhages or inflammation

as the inciting factor. One other congenital anomaly has always been present in every eye with dysplasia seen by me, and that is a persistence of the hyaloid artery, much more marked than the remnant occasionally seen. These vessels do not extend to or from the area of involvement but take the course of the hyaloid artery from the papilla to the posterior surface of the lens. It may be that because of fetal inflammation of the choroid or retina excessive vascularization has taken place or the usual atrophy of the hyaloid artery has been retarded



A B

Fig. 4.—*A*, macular dysplasia or coloboma with a persistent hyaloid artery (seen indistinctly in the disk); *B*, an anterior polar cataract (in the same eye).

A B

Fig. 5.—*A* and *B*, macular dysplasia or coloboma, showing the curved hyaloid arteries as they pass from the disk to the lens.

because of the need of a greater blood supply to combat the inflammation or repair the damage. On the other hand, there may have been an arrest at a certain time of fetal development which involved the intrauterine vascular system as well as the local area in question (fig. 5).

As stated before, the fetal fissure extends from the margin of the cup to the forebrain along the ventral aspect of the optic cup and stalk. Thus, in the stalk there is also an invagination of the ventral wall. When the fissure of the stalk closes there is formed a double tube, one

within the other. The inner tube, which consisted of the ventral wall of the stalk, is soon invaded by the axons from the ganglion cells of the retina in the formation of the optic nerve, and it disappears or forms supporting tissue for these nerve fibers, which completely fill the stalk by the middle of the second month of gestation.

The nerve fibers begin to invade and replace the inner layer of the optic stalk, leaving only a remnant or plug at the papillary end called the primitive epithelial papilla, which forms a cone of glial tissue known as Bergmeister's papilla. If this condition persists, the term gliosis or pseudoneuritis is applied to it. Later a sheath is formed from this glial tissue about the hyaloid artery for a third of the distance through the vitreous. This sheath, with the passing of the hyaloid artery, atrophies as far back as the papilla (about the seventh month), and the disk shows, depending on the amount of its recession, a deep or shallow physiologic cupping (fig. 6 *A*).

A B

Fig. 6.—*A*, persistent Bergmeister's papilla. *B*, coloboma of the optic nerve.

The outer layer of the optic stalk, which is nonpigmented in man, becomes converted into the neuroglial supporting tissue of the optic nerve.

Certain anomalies are found in the development of the optic nerve. Colobomas occur which are due to nonclosure of the fetal fissure (fig. 6 *B*). These are of various types, depending on whether they involve just the nerve or the nerve with the adjacent choroid. Inferior conus is probably a slight coloboma of the interior marginal zone between the choroid and the papilla. In this condition the pigment of the outer layer of the retina does not reach the optic stalk, resulting in the arrest of development of the choroid, pigment layer and retina, except the nerve fiber layer, below the optic papilla (fig. 7 *A*). Other congenital conditions, such as cysts and holes, are found in the papilla.

The surface ectoderm is stimulated to growth by the approach and contact of the optic vesicle. A thickening of the ectoderm, the lens

plate, appears, which pits and forms the lens vesicle. As the optic cup invaginates, receding from the surface contact, the lens vesicle develops and follows it, filling the space between the margins of the cup. Adhesions form between the receding retinal layer of the optic cup and the advancing posterior surface of the lens vesicle, and as the lens does not keep up with the invaginating retina, the adhesions stretch into a fibrous scaffolding on which the primary vitreous forms. Until the second month there is protoplasmic continuity between the lens and the adjacent structures. After this the hyaline capsule appears, probably secreted by the cells of the lens vesicle, and the lens becomes independent.

In order for the lens to begin development, it seems necessary for the surface ectoderm to be in contact with the optic vesicle. If this contact is not made, there is no trace of a lens. However, congenital

Fig. 7.—Inferior conus or crescent. There was also a coloboma of the upper lid in this case. *A* shows the section of the eye (supplied by Dr. M. K. Asbury), and *B*, the appearance of the eyelid.

aphakia is rare. If there is an optic vesicle, a lens of some kind is usually present, though it may be rudimentary, as in the case of microphakia found most frequently in microphthalmic eyes.

As the lens vesicle leaves the surface, a lens stalk forms, uniting the two. The vesicle soon separates (fourth week), the lens stalk disappears, the surface ectoderm unites and mesodermal tissue pushes in between. If there is a delay in the separation of the lens vesicle, certain types of anterior polar cataracts or lenticonus may form (fig. 4 *B*).

At about the time of lens encapsulation the cells of the posterior part of the lens vesicle elongate, growing forward until, by the end of the second month, the vesicle is practically filled. If these fibers fail to develop, the so-called "disk-shaped" cataract forms. As there is no

embryonic nucleus present to support the fibers developing from the equator, a flat disk-shaped or doughnut-shaped lens results.

Shortly after the lens vesicle has been filled by the fibers from the cells of the posterior part of the vesicles, the cells of the equator develop fibers which pass forward and backward toward the poles. As these fibers are not of the same length, they do not meet at a point but form suture lines. These suture lines occur over the clear embryonic nucleus formed by the posterior lens fibers. The first are a simple anterior horizontal and a posterior vertical suture which, as more fibers are laid on them, change into an anterior erect and a posterior inverted Y (fetal nucleus developing from the third to the eighth month). As more fibers develop, the suture pattern becomes more arborescent and complicated.

Absence of the formation of the equatorial fibers results in the formation of the congenital morgagnian cataract. This cataract, however, may be due to degeneration and not to an arrest in growth, as a full-sized lens capsule is usually present which contains a milky fluid in which is found the embryonic nucleus.

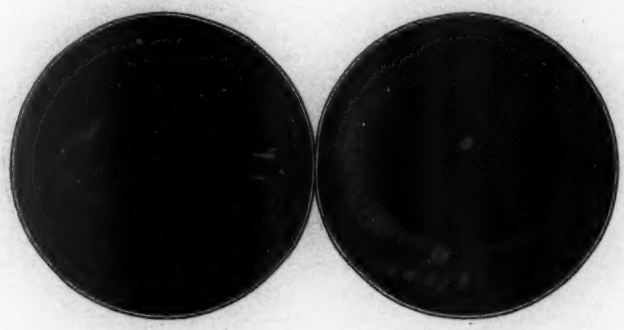
Beside the two aforementioned cataracts, there are probably a few other types of cataract due to arrested development. At the posterior pole of the lens, residual mesodermal tissue, remnants of the perilenticular vascular capsule, may be found from which hangs a vestige of the hyaloid artery. Posterior polar and capsular cataracts may result from the presence or persistence of these tissues. Anterior polar cataract may form due to an arrest in the recession of the pupillary membrane or in the imperfect separation of the lens stalk at an early period. Other opacities of the lens which are termed congenital cataracts are probably not due to simple arrested development and are not a part of this discussion.

Adhesions formed between the lens and the ciliary process when they are in close proximity (fifth month) may lead to dislocation or coloboma of the lens. The suspensory ligaments of the lens are probably formed from fibrous structures spanning the gap between the equator of the lens and the neural ectoderm of the optic cup. Fibers may be derived from the ciliary region and, with the growth of the tertiary vitreous, extend to the lens capsule. Ectopia lentis and coloboma lentis may be due to absence, underdevelopment or yielding of these suspensory fibers (fig. 8).

The margins of the optic cup begin active growth at about the third month. The inner layer of the ciliary process begins to fold. The edge of the cup develops into the epithelial iris. The two layers of the ciliary process adhere. Arrest in development of the epithelial part of the iris may result in aniridia or coloboma of the iris. The fetal fissure

is normally closed long before the development of the ectodermal iris; therefore it plays only a secondary part in the formation of a typical coloboma of the iris and no part in the formation of an atypical coloboma. Atypical coloboma of the iris and ciliary body may be due to arrest or delay in the waves of extension of the margin of the cup which never catch up with the normal advance. Some authors believe that mesodermal tissue or blood vessels check the normal progress, causing coloboma. Others believe that this mesodermal tissue is secondarily found trying to fill in the defect but has no primary importance, as it does not offer enough resistance for interference. Factors causing coloboma of the iris and ciliary body may secondarily cause coloboma or luxation of the lens by interference with the normal development of the suspensory ligaments.

A small space at the margin of the optic cup, a remnant of the primary optic vesicle, begins enlarging about the third month along



A B

Fig. 8.—Ectopia lentis, in *A* with choroideremia and in *B* with aniridia.

with the active growth of the developing iris. After the fifth month this space decreases in size, to disappear by the seventh month. If there is an arrest before the stage of shrinkage, this space may remain or may dilate to form cysts of the iris or "grape seed" bodies, so named because of the pigmented walls.

The ectodermal part of the iris is composed of a pigmented posterior and an unpigmented anterior layer. The sphincter muscle develops from the anterior layer at about the fourth month and the dilator muscle at about the sixth month. Arrest in the development of the dilator muscle of the iris results in congenital miosis, as the sphincter, which develops first, is unopposed. If the sphincter does not develop, the dilator muscle does not form.

The retina begins differentiation at the beginning of invagination of the optic cup. It reaches its adult thickness by the middle of the second month. In developing, the layers separate from within outward in the

opposite direction to the functioning nerve impulse. The macular area begins developing about the third month but is not completed until several months after birth. The remaining retina is about completely developed by the eighth month. Arrest in the development of various layers of the retina probably accounts for a number of congenital or early changes which are called infantile degenerations, caused by the failure of the inner wall of the optic cup to complete its differentiation.

There are also a number of postnatal anomalies of the fundus which are developmental conditions of familial and hereditary background. These conditions are not thoroughly understood. All show an early abiotrophy or premature degeneration due to an inherent lack of durability. A few of the more common conditions are mentioned.

Leber's hereditary atrophy of the optic nerve is germinal in origin. It shows a failure of development, or atrophy, of the papillomacular bundle in both eyes. Central scotoma is present. This is a markedly

A B

Fig. 9.—*A*, Leber's hereditary atrophy of the optic nerve, due to failure of development or atrophy of the papillomacular bundle. The disk and retina show atrophy of the nerve fibers. *B*, congenital retinitis pigmentosa, due to regional failure of development of both ectoderm and mesoderm.

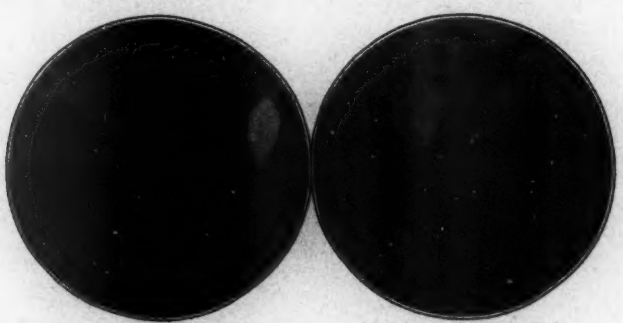
hereditary condition. It is sex linked, affecting the males through the unaffected females (fig. 9*A*).

In Tay-Sachs amaurotic family idiocy there is an almost complete absence of the nerve fiber layer of the retina. The ganglion cells show some degeneration. The optic disk is atrophic. This seems to be purely an ectodermal failure and is typically abiotrophic (fig. 10).

There are a group of conditions that appear to be due to a failure of both ectoderm and mesoderm though one usually predominates. In retinitis pigmentosa the visual elements may be primarily affected or may atrophy because of deficient choroidal development or sclerosis, which denies proper blood supply to the retina. Involvement is seen first at the equatorial region with a ring scotoma, which may enlarge peripherally and then centrally. Pigment, the shape of bone corpuscles, is present. The retinal arteries are constricted, and the disk is pale. Night blindness

is present. This may be a dominant, recessive or sex-linked condition (fig. 9 *B*).

In choroideremia there is a lack of pigment, so that the fundus is white. This may be due to the failure of the ciliary arteries (short posterior and anterior) to bud from the ophthalmic artery at an early stage of development, or it may be due primarily to a still earlier failure of pigment formation in the outer wall of the optic cup.



A B

Fig. 10.—*A* and *B*, Tay-Sachs amaurotic family idiocy. In this condition there are absence of the nerve fibers of the retina and some degeneration and loss of ganglion cells, due to failure of ectodermal development. (Photographs of both retinas of Dr. A. Levin's patient.)

A B

Fig. 11.—*A*, choroideremia. In this condition there is absence of pigment in the fundus due to failure of ectodermal or mesodermal development. *B*, gyrate atrophy of the choroid and retina. This condition is similar to that shown in *A* but is not so extensive.

An allied condition is that of gyrate atrophy of the choroid and retina in which there are gradually enlarging round or oval patches due to loss of choroid and retina. Figure 11 shows photographs of the fundi of cousins whose kinship is from the marriage of daughters of two brothers to the two sons of the third brother. One fundus shows gyrate atrophy of the choroid and retina and the other choroideremia.

Both boys suffered from night blindness and had constricted fields with poor vision.

Anencephaly should probably be mentioned. In this condition there is an absence of nerve fibers, and the ganglion cell layer is frequently involved. There is a failure of development of the whole brain above the medulla. The cause is theoretic but is possibly due to an embryonic hydrocephalus. There seems to be a normal development of the brain and eye to the middle of the second month with subsequent degeneration of brain and optic nerves (fig. 12).

Glioma or neuroblastoma has a place in this discussion, as the malignant growth of cells of the nuclear layers of the retina occurs after an arrest or undue persistence of nondifferentiated cells of the inner layer of the optic cup, which then begin to divide excessively at too late a

Fig. 12.—Anencephaly. In this condition there is absence, probably degeneration, of the nerve fibers with involvement of the ganglion cell layer.

period. Such conditions are seen as a recessive or irregular dominant type of heredity.

ARREST IN THE DEVELOPMENT OF MESODERMAL STRUCTURES

Paraxial mesoderm surrounds the optic vesicles during the early stages of development, invading the space between the surface ectoderm and the lens and providing the mesodermal tissue of the eye and orbit. The substrate of mesoderm appears to have a part in the arrest of proper development, leading to the formation of the cyclopean eye. There may be a lack of paraxial mesoderm or an excess of visceral mesoderm which prevents the lateral extension of the optic vesicles, though the condition is probably primarily due to an arrest in the development of the ectodermal structures (fig. 2).

The visceral mesoderm (maxillary process) develops from below laterally and helps to rotate the eyes from a lateral 160 degree angle to a forward 62 degree position. In oxycephaly there is a failure of proper development of the visceral mesoderm. The lower part of the skull and adjacent structures fail to expand due to the premature synostosis of the transverse sutures. The eyes are placed far apart, as the normal action of the (arrested) tissues to shift their position is not available. The orbits are shallow. Atrophy of the optic nerve is not infrequent (fig. 13).

Both the paraxial and the visceral mesoderm, but especially the former, are vessel forming as well as tissue forming. The visceral meso-

Fig. 13.—Oxycephaly. There are normal development of the paraxial mesoderm and arrested development of the visceral mesoderm. The orbits are wide apart and shallow.

derm forms the lower and outer wall of the orbit and the tissue of the lower lid. The paraxial mesoderm provides the remaining mesodermal structures of the eye and orbit.

Blood channels develop in the form of a plexus by outbudding from the carotid artery and extend along the lower surface of the optic stalk and vesicle. The chief vessel is the hyaloid, which lies in the fetal fissure at first. It extends forward to the posterior part of the lens, where it invades the fibrous perilenticular capsule and forms the vascular tunic. Other branches from the plexus pass along the surface margins of the fissure and upward along the edge of the cup on either side to meet above, forming the annular vessel. Later the hyaloid (internal ophthalmic) artery, before entering the stalk, gives off the common

ciliary (temporal) and nasal ciliary arteries. The final formation is the nasal artery and the two long ciliary arteries, which anastomose to form the major circle of the iris. The branches of the long ciliary arteries form the posterior ciliary arteries.

The venous system begins as blood spaces, which coalesce to form veins. The drainage from the eye is by way of the venae vorticosae (later the superior and inferior ophthalmic veins) into the superior and infraorbital plexuses, thence into the cavernous sinus.

Blood channels form on the surface of the vesicle when pigment appears in its walls, forming the primitive choroid. This network of surface vessels, the choriocapillaries, connect anteriorly with the upward growing vessels at the margin of the cup which meet to form the annular vessel and which have been joined from within the cup by branches from the hyaloid system.

As the intraocular vascular system develops, buds arise from the main trunk of the hyaloid artery and ramify throughout the (primary) vitreous. The vessels thus formed are called the vasa hyaloidea propria. The posterior vascular tunic of the lens develops as the branches from the hyaloid artery ramify through the fibrous perilenticular capsule which has just formed around the lens, probably from mesodermal and ectodermal tissues. These vessels communicate with the extraocular vascular system by passing between the lens equator and the rim of the cup to anastomose with the annular vessel.

Some authors believe that arrest of normal growth at the rim of the cup and at the lens equator, resulting in coloboma of these structures, is due to the presence of these communicating vessels, which offer an obstacle to the developing ectoderm. Others believe that no resistance is offered and that the mesodermal tissue which does not atrophy is pushed aside and plays no part in the arrest of ectodermal growth in this area.

The mesoderm anterior to the lens is vascularized about the second month by off-shoots from the annular vessel.

The anterior mesodermal structures of the eye are formed from an invasion of the paraxial mesoderm between the lens and the surface ectoderm immediately after their separation. Soon vacuoles form, splitting the mesoderm into two layers. The external layer remains in contact with the surface ectoderm to form the substantia propria of the cornea. If this formation is defective in its center, keratoconus or keratoglobus may result. If it does not develop in its proper secondary radius of curvature, a congenitally flat cornea remains. Megalocornea is an early and microcornea a late arrest in corneal development.

The internal layer of this mesoderm cleavage remains in proximity to the lens and forms the stroma of the iris. As this mesodermal tissue appears much earlier than the ectodermal tissue which goes to form the

iris, it possibly acts as a scaffolding on which the ectodermal iris develops. Whether aniridia is due to an arrest in the developing ectoderm at the margin of the cup primarily or whether it has been suppressed or misdirected by the mesoderm which precedes it has not been definitely determined (fig. 8 *B*).

Later, the central pupillary portion of the vascularized anterior lens tunic undergoes atrophy. Arrests in the formation of the pupil result in a pupillary membrane which may be just a few strands stretching between the lesser circles of the iris or a considerable amount of stromal tissue consisting of a meshwork containing starlike cells with occasional vascularization and pigmentation. Inflammation during development may have caused it to become attached to the anterior surface of the lens, forming an anterior polar or capsular cataract (fig. 4 *B*).

Beginning about the third month the ciliary muscle is formed from the paraxial mesoderm at the outer edge of the cup. The mesodermal muscular portion is completed by birth. The circular muscular portion continues to develop after birth in direct proportion to the amount of the hypermetropia and inversely to the size of the globe. An arrest in development of the eye as a whole would lead to congenital hypermetropia.

The choriocapillaries develop in the early condensation of paraxial mesoderm about the optic cup. It has been stated previously that interference with their development may lead to coloboma of the retina (choroid). Later, the outer layer of choroidal vessels which lead to the venous plexuses form. During the fourth month the middle layer of the choroidal vessels, which is arterial, is formed from the ciliary arteries as they break up to form the choriocapillaries.

The scleral condensation, which began first in the areas of condensation for the insertion of the extrinsic ocular muscles, proceeds forward and backward. If there is an arrest in its development, causing a thinning of the sclerotic coat, congenital myopia or posterior staphyloma may result.

A cone of mesoderm forms about the optic cup and stalk at an early stage. This cone becomes segmented into the extrinsic muscles. The levator palpebrae muscle separates from the superior rectus muscle later. If this fails to happen, congenital ptosis results. Ptosis is related to epicanthus, which is normal in the human fetus and the mongolian adult. Both conditions alone are dominant, but together they may be either dominant or recessive. However, the recessive type is central in its cause, which produces an ophthalmoplegia, whereas the dominant type is due to an absence of the levator muscle.

During the last few months of fetal life the anterior segment of the eye undergoes its most active growth. When the structures in this area have developed, there is a recession or shrinkage of mesodermal tissue,

especially at the angle of the anterior chamber. The relation of the structures in this area have assumed their proper position some time after the fifth fetal month. If the mesoderm in the angle has been arrested or if the residual tissue blocks its capacity to function as a major intraocular drainage system, buphthalmia or congenital glaucoma may develop. Examination of buphthalmic eyes usually reveals an arrest in the absorption of mesoderm in the angle of the anterior chamber and an arrest in the development of the canal of Schlemm. The iris is atrophic, probably secondarily (fig. 3 *B*). The eye is enlarged, and the sclera is ectatic. Bullous keratitis (so-called) with staphyloma may be present. The retina and optic nerve are usually atrophic, with reduction or absence of the ganglion cells. The lens may also be flattened by the increased intraocular pressure.

Intraocular vascular retrogression or atrophy begins with the vasa hyaloidea propria about the third fetal month and proceeds forward, involving the posterior vascular lens capsule, then the lateral capsulopupillary vessels and, finally, the anterior or pupillary portion of the vascular lens capsule. This retrogression should be complete by birth. However, remnants of the main hyaloid artery are not infrequently seen hanging from the posterior lens surface. The shrinking branches of the vasa hyaloidea propria curl and disappear. Remnants seen in adult life are termed muscae volitantes. The proximal intramural portion of the hyaloid artery remains, and from its papillary end retinal vessels develop. These vascular buds form vessels in the nerve fiber layer surrounding the disk. From them other vessels proceed peripherally and deeper in the retinal layers.

As the hyaloid artery shrinks, it breaks in two. The part attached to the posterior perilenticular capsule hangs as a spiral, which may persist and is rather frequently seen in children. If the arrest is more extensive, opacities remain which result in posterior capsular or polar cataract.

The papillary end of the hyaloid artery usually disappears, but there is an occasional remnant remaining which may be surrounded by the arrested hyaloid sheath derived from Bergmeister's papilla, which has not regressed either.

The last of the intraocular vascular system to go are the vessels in the pupillary area, which, about the seventh month recede by arcades back to the lesser circle. As previously stated, arrests in this regression may result in the formation of a pupillary membrane and opacities of the anterior lens capsule.

SUMMARY

Some of the more common and most discussed conditions of arrested development of the eye which are of interest to the practicing ophthalmologist are presented as they are met with from time to time and must be considered in his diagnosis and treatment.

News and Notes

SOCIETY NEWS

New Haven Ophthalmological Society.—The first meeting of the newly formed New Haven Ophthalmological Society was held on April 17, 1939. The officers of this society will be found in the Directory of Ophthalmologic Societies on page 1085 of this issue.

UNIVERSITY NEWS

Graduate Course in Visual Optics and Physiology at Harvard Medical School.—The Harvard Medical School will offer a graduate course in visual optics and physiology from July 1 to 29 inclusive. The course is designed for those who desire to teach or to do research work in physiologic optics. Instruction will include lectures and laboratory work on reflection, refraction, refractive errors of the eye, accommodation, ophthalmoscopy, retinoscopy, reduced eye, aberrations of the eye, ocular motility, binocular acuity, light sense, color vision and color blindness, entoptic phenomena, impulses of the optic nerve, visual illusions, pupillary reflexes and intraocular pressure. Instruction will be from 9 a. m. to 5 p. m. daily. Attendance is limited to eight. The fee for the course is $150.

CORRECTION

In the article by Dr. T. J. Dimitry entitled "Evolution of a Sucking Disk for Intracapsular Extraction of Cataract," in the February issue (ARCH. OPHTH. **21:** 261, 1939), the sentence at the bottom of page 262 should read as follows: "The three parts of the outfit must be disassembled and may then be sterilized by boiling or by immersion in alcohol."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Color Sense

INFLUENCE OF SOUND IRRITATION ON THE COLOR SENSE OF A PROTANOPE. S. V. KRAVKOV, Vestnik oftal. **11:** 103, 1937.

Kravkov made a number of experiments on a protanope in an effort to establish the relation between sound and variation of color sense. Nagel's anomaloscope and Ishihara's tables were used; the irritator was the sound from a lamp sound generator which was transmitted by telephone receivers to each ear. Two drawings and a table illustrate the dark adaptation curves and the color sensitivity with and without sound irritation.

Kravkov's conclusions follow:

1. The increase of color sensitivity in a protanope under the influence of sound is noticeable only in that part of the spectrum which touches the blue-sensitive apparatus of the eye (after the theory of Helmholtz).

2. The absence of change of the color sense in the rest of the spectrum speaks in favor of the red-sensitive and the green-sensitive apparatus not existing separately in the protanope; i. e., the curve of the "red" basic stimulation coincides with the curve of the "green" basic stimulation.

3. These experimental data confirm the previous findings that the green-sensitive and the blue-sensitive apparatus of the eye react to the sound stimulus by an increase of excitability, while the red-sensitive apparatus reacts by a decrease.

4. The color sense to the orange-yellow rays of the spectrum does not react to the sound stimulus. This fact contradicts Hering's theory of the presence of an independent yellow-sensitive substance of the color vision.

O. SITCHEVSKA.

Conjunctiva

A NEW FORM OF CONJUNCTIVAL MYCOSIS DUE TO A MOLD OF THE TYPE "GEOTRICHOIDES." T. MAESTRO, Ann. di ottal. e clin. ocul. **66:** 439 (June) 1938.

A housewife, aged 30, had noted signs of conjunctival irritation in the left eye and the presence of a small black spot on the bulbar conjunctiva for about a year. The spot measured 8 by 5 mm. and was distinctly black. Dark colored threads of mucus were present in the sac. The dark patch could be wiped off easily, leaving a small roughened area where the conjunctival epithelium was eroded. The black patch always reformed within four or five days. Many methods of treatment were employed, including large doses of the iodides by mouth and arsphenamine, but the lesion continued to recur. It finally disappeared

after a course of intravenous injections of the following preparation: metallic iodine, 10 Gm.; potassium iodide, 20 Gm., and water, 100 cc. This was diluted with a double quantity of a 20 per cent solution of sodium hyposulfite just before injection. The initial dose consisted of 0.5 cc. of the iodine solution, which was increased to 1 cc. and 2 cc., the succeeding doses being 2 cc. Three injections a week were given for a total of ten injections, after which symptoms of intolerance were observed. During this time the conjunctival lesion was treated with a 2 per cent solution of potassium iodide, given by iontophoresis with a current of 3 milliamperes for fifteen minutes, the treatment being given every second day. After twenty days the lesion disappeared and had not recurred after three months. Cultures and smears showed a mold, which could be classified as of the type Geotrichoides. Animal inoculations were negative, but the organism was found in three of four successive cultures and was never found on the normal conjunctiva. It grew readily on Sabouraud's medium and on other mediums.

Full bacteriologic details and photomicrographs accompany the article.

S. R. GIFFORD.

EXISTENCE OF BITOT SPOTS WITHOUT ANY CONNECTION WITH XEROPHTHALMIA. SIE BOEN LIAN, Arch. f. Ophth. **139:** 17 (Aug.) 1938.

Persons with Bitot spots in whom vitamin A deficiency can be ruled out are encountered fairly commonly in Batavia. From Mulock Houwer's clinic in Batavia the author reports 19 cases of Bitot spots in which the dark adaptation was normal, with the exception of 1 case in which night blindness did not improve despite large doses of cod liver oil given over long periods. The author determined the amount of carotenoids and vitamin A in the serum in these 19 cases and found it to be high in 9 and slightly below normal in the rest. The cod liver oil did not seem to have any influence on the Bitot spots, which either remained stationary for several years or disappeared and reappeared without apparent reason. The pigmentation of the inferior fornix and the plica semilunaris, which Pillat described as a sign of vitamin A deficiency, was absent in all cases. Histologically, these Bitot spots presented themselves as patches of thickened, keratosed epithelium which had also undergone fatty degeneration. The cause of Bitot spots which are not associated with xerophthalmia is unknown.

P. C. KRONFELD.

Cornea and Sclera

CORNEAL TRANSPLANTATION: RESULTS OF A SERIES OF FIFTY-SIX OPERATIONS ON FORTY-EIGHT EYES. J. W. TUDOR THOMAS, Brit. M. J. **2:** 740 (Oct. 8) 1938.

Since his last paper, Thomas has modified his operation for corneal transplantation by using saline solution instead of oil to receive the graft and by arranging the cross stitches so that the knots do not lie on the graft or on the margin. The adoption of these modifications seems to have been followed by improved results.

The results of operation should be judged by the clarity of the graft, any membrane that may have formed on its posterior surface being taken into account. It is interesting to note that the improvement after corneal grafting was maintained in 23 of 30 eyes over a period of some years. The author reports that 62.57 per cent of all the operations performed were successful, that successful results were obtained in 9 cases of interstitial keratitis and that 92 per cent of operations in cases in which conditions were favorable were successful.

It is now believed that the modified technic will reduce the deterioration of the grafts over a period of years. The best visual result obtained was 6/9, and some patients obtained vision of 6/12.

ARNOLD KNAPP.

CORNEAL ULCERATION AND VITAMIN A DEFICIENCY. HERBERT WOLFF, Acta ophth. **16:** 323, 1938.

The case reported is that of a man of 38 with a marginal corneal ulceration which resisted the usual therapeutic procedures. The patient had brittle finger nails, and when he was tested with Gullstrand's photometer, marked night blindness was detected. Both the corneal lesions and the night blindness cleared after a course of parenteral injections of a vitamin A preparation. Vitamin A by mouth was not effective in controlling the pathologic changes in the cornea.

The vitamin deficiency in this case was not due to any dietary lack and probably not to faulty intestinal absorption. The author believes some obscure disturbance in metabolism of the liver is responsible for the faulty utilization of vitamin A.

O. P. PERKINS.

Experimental Pathology

EXPERIMENTAL MYCOSES OF THE LENS. M. MECCA, Ann. di ottal. e clin. ocul. **66:** 481 (July) 1938.

The literature on experimental ocular infections, especially the few articles dealing with infections due to members of the Trichophyton group, is reviewed. Mecca employed a suspension of the spores of Trichophyton gypseum. In a first group of experiments inoculation was made directly into the rabbit's lens. In a typical example the postoperative reaction subsided after four days, and multiplication of the mold could be seen by an increase in the zone of opacity and the presence of fine yellow threads in the lens. After twenty days the mold had extended through the whole anterior cortex and into the nucleus and the posterior cortex. After forty-five days, when the whole lens appeared to be invaded, signs of iritis appeared, and soon after the pupil was occluded by exudate. Sections showed mycelia in the cortex of the lens, the nucleus and subcapsular zones being left relatively intact. There was infiltration of the anterior segment with lymphocytes and plasma cells, but no threads were found outside the lens. In a second group of rabbits discission of the lens was performed, and six hours later the anterior chamber was inoculated with spores. Elements of the mold developed only in the portions of the lens remaining within the capsule, none being found in the anterior chamber. Inflammation of

the anterior segment more intense than that in the first experiments resulted in all the animals. In a third group of rabbits the emulsion of spores was injected intracardially, followed by discission of the lens and laceration of the iris. No mycelial elements were found in the eyes of these animals, which were killed after various periods. In a fourth group of rabbits, cultures of the mold were made in Sabouraud's medium to which rabbit or beef lens had been added and also from the lenses inoculated in the preceding experiments. These cultures were positive with lens material from animals of the first and second groups but not with that from animals of the third group.

A bibliography and photomicrographs accompany the article.

S. R. GIFFORD.

CHANGES IN THE RETINA PRODUCED BY INJECTIONS OF EPINEPHRINE HYDROCHLORIDE INTO THE VITREOUS. M. MECCA, Ann. di ottal. e clin. ocul. **66:** 561 (Aug.) 1938.

Mecca reviews the reports dealing with the production of retinal changes by injections of epinephrine hydrochloride into the vitreous and with the production of a condition resembling retinitis pigmentosa both by repeated injections into the vitreous of epinephrine hydrochloride alone and by this procedure followed by irradiation of the eye with light of a wavelength of from 380 to 433 millimicrons. He injected 0.2 cc. of a solution of epinephrine hydrochloride (1 : 1,000) only once into the vitreous of rabbits, watched the fundi carefully and sectioned the eyes after varying periods. In a group of rabbits killed after five days the picture of marked arterial spasm was observed. The vessels became smaller in the days after the injection, and edema of the retina, appearing on the second day, was still increasing on the fifth day. Sections showed the edema to be chiefly in the nerve fiber layer, the retinal arteries being small and free from red cells. The choroidal vessels were not affected or congested. In a group of rabbits killed after fifteen days the edema had begun to subside, and on the eighth day fine brown granules were seen in the retina, which increased in number during the following days. Sections showed the pigmented cells to have migrated from the pigment epithelium. There was marked degeneration of the rods and cones, and the retina was adherent to the choroid. In a group of rabbits killed after a month more pigmentation had developed and the arterial spasm was still present. Sections showed the disappearance of the rods and cones and of the inner granular layer, while pigmented cells were present in masses throughout all layers of the retina. The pigment epithelium was irregular and rarefied in areas corresponding to the retinal pigmentation. In animals killed after two months the same picture was present, except that the pigment increased in the equatorial region, reaching almost to the periphery. After four months the retina was adherent to the choroid in many areas and was invaded by processes of choroidal cells. The choroid was now markedly atrophic. No changes were seen in a group of animals that received injections of physiologic solution of sodium chloride into the vitreous, and injection of epinephrine hydrochloride into the vitreous of dogs produced only a transient arterial spasm, which disappeared after from ten to

thirty minutes. In albino rabbits the picture of vascular spasm and edema was produced, but no pigmentation occurred.

A bibliography and photomicrographs are included.

S. R. GIFFORD.

General

FURTHER RESEARCH ON EYES OF MONOZYGOTIC TWINS OF ADVANCED AGE WITH REFERENCE TO THE HEREDITY OF SENILE SYMPTOMS. A. VOGT, Klin. Monatsbl. f. Augenh. **100:** 497 (April) 1938.

Vogt calls attention to a previous series of studies on determination of senile changes in advanced age, predestined in the germ plasma, which he demonstrated in monozygotic twins, aged 55 to 81. The identical appearance in both twins of furrows in the skin, baldness, graying of the hair and mustache, hair on the chin of old women and other senile changes showed that their occurrence was independent of outside influences but dependent on the formation of the germ plasma. The absence of these senile symptoms and of opacities of the lens was due to the same cause. In the present paper Vogt publishes his observations of identical changes in the bulbar conjunctiva (pinguecula), cornea (gerontoxon, pterygium and conditions of the corneal endothelium), iris, lens, vitreous and posterior segment of the eyeball, including the disk and surrounding area. Twenty-three senile and presenile identical twins were studied. The paper contains 123 illustrations, many of which are colored; pictures showing the macroscopic, histologic and slit lamp appearance of the eyes and the photographs of about 20 pair of twins are included. The divers forms of senile cataract receive detailed consideration in the text and the illustrations. Vogt demonstrates the hereditary transmission of Krukenberg's pigmented spindle, cornea guttata senilis and senile degeneration of the pigment edge of the pupil.

Vogt states definitely that medication is futile. There is a special toxin for each hereditary form of cataract, hence a special remedy would be required for each form. Studies of the chemistry of the lens are bound to yield as little result, in Vogt's opinion, as an attempt to prevent graying of the hair or as chemical analysis of the optic nerve in Leber's atrophy of the optic nerve. Quotations of recent publication by such internists as Naegeli, Bergmann and Thannhauser support the author's view.

The hereditary anlage is decisive for life not only from birth to full growth of the body but in middle age, and it finally determines the symptoms in senescence which precede the physiologic termination of life.

K. L. STOLL.

General Diseases

OCULAR COMPLICATIONS IN THE COURSE OF SPIROCHETAL HEMORRHAGIC ICTERUS. M. L. GUILLAUMAT, Bull. Soc. d'opht. de Paris **50:** 314 (June) 1938.

Ocular complications in the course of spirochetal hemorrhagic icterus are well known but have rarely been observed in Paris. A traveler,

aged 37, complained of *mouches volantes*. The vision in the left eye was reduced to 0.9, while that in the right eye was normal. The lens and vitreous of the left eye were entirely transparent. However, the optic disk was hyperemic, and its edges showed some edema. The macula, visual fields and tension were normal. A history of a previous acute febrile attack of hemorrhagic icterus was elicited. A mercuric preparation was administered intravenously. Later there was pericorneal injection of the left eye. The iris was muggy and dilated poorly, some posterior synechiae preventing good dilatation. Vision was reduced to 1/20. After a subconjunctival injection of epinephrine hydrochloride the iris dilated well. A small hypopyon was noted in the anterior chamber. Recovery was extremely slow. The right eye remained quiet except for a few precipitates on the posterior corneal surface. The Wassermann reaction of the blood was negative. No tests were made to find spirochetes in the urine or in the spinal fluid. This is regretted by the author. The border of the iris of the left eye remained irregular, but the visual acuity returned to 0.9.

L. L. MAYER.

OCULAR COMPLICATIONS OF INTESTINAL AMEBIASIS. M. BAQUIS, Rassegna ital. d'ottal. **7:** 338 (May-June) 1938.

A man, aged 31, noted diminution of vision eight months after an attack of dysentery. Vision in the left eye was reduced to 1/50, owing to a large central area of chorioretinitis. Some opacities of the vitreous were present. The right eye, though having normal vision, showed several small foci of active choroiditis. Amoeba histolytica was present in the stools. The results of physical examination were otherwise negative. After treatment with emetine hydrochloride the stools became negative, and vision improved to 7/10, while the chorioretinal lesion healed with moderate scarring. After the condition remained quiescent for a year, fresh foci of choroiditis developed. Although the stools remained negative, an abscess of the liver was suspected.

S. R. GIFFORD.

MYELOGENOUS CHLOROMA. A. A. LEBEDIEV, Vestnik oftal. **11:** 195, 1937.

Lebediev states that this is the eighty-first case of myelogenous chloroma reported in the literature. A boy, aged 13 years, presented bilateral exophthalmos with bilateral papilledema, more marked on the right side. There were paralysis of the facial nerve on the right side, lagopththalmos and superficial keratitis in the right eye. A firm, immovable tumor could be palpated at the outer upper third of the orbit. The blood picture indicated a disease of the blood-forming organs, characteristic of a neutrophilic leukocytosis. Autopsy showed a chloroma in each orbit, numerous metastases in the occipital and temporal bones, in the aponeurosis and intercostal muscles and hyperplasia of the brain. The growths were greenish. There was general hyperplastic lymphad-

enitis, the lymph nodes having a greenish color on cross section. The clinical differential diagnosis, treatment and prognosis are discussed.

The conclusions are as follows:

1. Chloroma belongs to one of the forms of leukemia.
2. The diagnosis of chloroma in some cases is difficult, particularly in the absence of the characteristic color.
3. Roentgen examination does not always aid in making the diagnosis.
4. Biopsy is valuable for early diagnosis.
5. Roentgen therapy is the only logical treatment at present.
6. Each case of chloroma should be reported in order that more material may be obtained for the study of the etiology, early diagnosis and treatment of the disease.

O. SITCHEVSKA.

General Pathology

THE QUESTION OF SECRETORY PROCESSES OF THE PIGMENT EPITHELIUM. S. CIOTOLA, Ann. di ottal. e clin. ocul. **66:** 612 (Aug.) 1938.

Stimulated by the reports of Koyanagi on the part played by the apparent secretion of the pigment epithelium in various pathologic conditions, the author studied the changes in this layer in eyes removed because of Coats's retinitis, sympathetic ophthalmia, exudative choroiditis, glioma and retinal and metastatic carcinoma of the choroid. In eyes with Coats's retinitis externa droplets taking the fat stains were found in the pigment epithelium, and the subretinal exudate stained with sudan III and could be seen in direct continuity with the contents of the pigment epithelium. Some of the epithelial cells were swollen and globular and contained large amounts of lipoid granules. Specimens from 3 persons with this condition were examined. Similar changes in the pigment epithelium were found in eyes with the other conditions associated with subretinal exudate. The author cannot decide whether the part played by the pigment epithelium in the formation of the subretinal fluid is secretory or whether it simply modifies in some way the fluid originating in the choroid, making it different from an ordinary transudate.

S. R. GIFFORD.

Glaucoma

STURGE-WEBER'S DISEASE, ESPECIALLY FROM THE POINT OF VIEW OF OPHTHALMOLOGIC SYMPTOMS. K. G. BENTSEN, E. DALSGAARD-NIELSEN and H. MÖLLER, Acta ophth. **16:** 279, 1938.

Naevus flammeus of the face, glaucoma, epileptiform seizures and angiomatous changes in the cerebral cortex, particularly in the occipital lobe, make up a syndrome which has been referred to of late as Sturge-Weber's disease.

The vascular formations in the brain, if calcified, produce characteristic changes in the roentgenogram and are also likely to produce a hemianopic defect of the visual field in addition to other neurologic disturbances. Choroidal angiomas may occur and in turn may produce

glaucoma or an ophthalmoscopic picture resembling that of exudative retinitis.

All of these signs and symptoms as well as others are exemplified in this article in the report of 3 cases. O. P. PERKINS.

Hygiene, Sociology, Education and History

PRESCRIBING LIGHT: AN IMPORTANT FACTOR IN THE CARE AND TREATMENT OF THE EYE. C. E. FERREE and G. RAND, Brit. J. Ophth. **22**: 641 (Nov.) 1938.

The subject is treated of under the headings of "Important Structural and Functional Considerations," "Outstanding Features in Lighting in Relation to the Examination and Care of the Eye," "Means of Improving Lighting Conditions," "Examination of the Eye in Relation to Lighting," "Intensity of Light and Strength of Reading Glasses" and "Summary."

This lengthy article contains new material and the substance of many of the authors' previous communications. The authors state that the results obtained and the principles discovered, however, belong essentially to the general subject of lighting. They are not sufficiently specific to be of direct and significant service to the physician. For him, the subject narrows down primarily to what is needed for the individual patient who comes under his care. W. ZENTMAYER.

Injuries

OCULAR INFECTION WITH GAS BACILLUS: REPORT OF CASES. G. CANON, Klin. Monatsbl. f. Augenh. **100**: 394 (March) 1938.

Infection of the eye with gas bacillus has its original focus in the vitreous and muscles, spreading to the fat and perimysium to a certain extent. The disease, which was known in the eighteenth century and was observed twice by Chaillous in 1904, was given greater consideration during the World War. Canon adds the reports of 6 cases of ocular infection with gas bacillus to the 22 cases described in the ophthalmologic literature of the world. Characteristic symptoms were present in all 6 cases, making early recognition possible. Perforation of the globe was the port of entrance in each case, and metallic foreign bodies caused the wound in all but 1 of the cases reported in the literature. Sufficient for the infection in some instances was the mere fact that the piece of metal came in contact with the eye without entering the globe. The first symptoms of severe infection developed after a short period of incubation of from four to twenty-one hours. Complete panophthalmitis was manifest after about eighteen to forty-three hours. An etiologic symptom was sanguinolent or lacquer-colored discharge with gas bubbles somewhere within the eyeball. The general health was good in some cases, while in others the picture of peritonitis was noted, depending on whether a genuine or a mixed infection was present. The disease is influenced by geographic, climatic and seasonal conditions. It predominates in the cold season during the months from

December to March. Districts with heavy loam are more favorable for the gas bacillus than those with sandy soil, and moist weather is more favorable than dryness.

Severe general infection and death are rare complications in ocular infection with gas bacillus, as it is limited to the eyeball. Specimen obtained by enucleation or evisceration showed the formation of a wide vascular zone in the choroid and copious accumulation of bacilli at a certain distance from the blood vessels. Anti-gas-edema serum acts prophylactically and furthers recovery.

K. L. STOLL.

Instruments

A FORCEPS FOR TOTAL EXTRACTION OF CATARACT. H. LAGRANGE, Bull. Soc. d'opht. de Paris **50:** 22 (Jan.) 1938.

The curvature of this new forceps coincides with that of the anterior surface of the lens. There are no teeth, but the inner side of each blade has a single long and deep serration. Photographs are included to demonstrate the application of the forceps. The shaft is angled backward as it reaches the blades, and thus manipulation is held to a minimum. Lagrange has had much success with the instrument and feels that fewer capsules are broken.

L. L. MAYER.

Lens

POSTOPERATIVE COMPLICATIONS OF CATARACT OPERATIONS. C. BERENS and D. W. BOGART, Am. J. Surg. **42:** 39 (Oct.) 1938.

Because many of the complications following operations for cataract may be prevented, Berens and Bogart analyzed the records of 1,004 extractions performed at the New York Eye and Ear Infirmary during the last two years. Extracapsular extractions were performed on 702 patients and intracapsular extractions on 302. The most frequent postoperative complications were hemorrhage into the anterior chamber (after 4 per cent of the intracapsular extractions and after 1.4 per cent of the extracapsular extractions) and loss of vitreous (after 9.9 per cent of the intracapsular extractions and after 9 per cent of the extracapsular extractions). The other complications, detachment of the retina, detachment of the choroid, suppurative intraocular infection and prolapse of the iris, occurred in only a few cases of each series. Secondary glaucoma occurred after 7 extracapsular extractions; iridocyclitis was observed following 7 extracapsular extractions and 2 intracapsular operations. If the number of postoperative complications is to be reduced, there seems to be little question that the preliminary examination is most important. Careful preoperative study of the patients, combined with the institution of treatment for chronic infections, hyperglycemia, hypertension and other general diseases, when found, may prevent serious postoperative complications which frequently result in impaired vision or blindness. Careful complete suturing of the wound with a complete but not too wide conjunctival flap seems to be one of the best methods of preventing serious postoperative complications. In the postoperative care of the patient the most important single factor in prevent-

ing complications (other than careful nursing and the treatment of cough and other symptoms causing movement which might endanger the eye) is the careful postoperative examination of the eye, especially the avoidance of squeezing by unnecessary or clumsy manipulation.

J. A. M. A.

Neurology

SYNDROME OF THE NASAL NERVE. C. DEJEAN and P. ARTIERES, Arch. Soc. d. sc. méd. et biol. de Montpellier **19:** 9, 1938.

Symptoms of neuritis of the nasal nerve were observed in a woman aged 66, who complained of sudden paroxysms of ocular pain on the left side which radiated to the zygoma, accompanied by transient desquamation of the corneal epithelium, marked lacrimation and profuse rhinorrhea. For the previous month one or more such attacks had occurred daily, lasting from two to three hours and being regularly associated with redness of the external bulbar conjunctiva. Vasomotor phenomena secondary to irritation of the nasal ganglion are probably the basis of the symptoms. In this case the left nasal fossa was painted twice at an interval of a week with a mixture of equal parts of cocaine, menthol and phenol, after which the manifestations gradually lessened and disappeared. The therapeutic test is considered to have confirmed the diagnosis.

J. E. LEBENSOHN.

ETIOLOGIC SIGNIFICANCE OF CALCIFICATION OR THROMBOSIS OF THE INTERNAL CAROTID ARTERY FOR VISUAL DISTURBANCES. P. SIEGERT, Arch. f. Ophth. **138:** 799 (July) 1938.

Roentgen examinations of the skull of elderly persons with or without visual disturbance reveal a high incidence of calcification of the circle of Willis. The presence of such a calcification in cases of lesions of the third or fourth neuron of the visual pathway does, therefore, not justify the conclusion that the lesion is caused by compression of the optic nerve or tract by the sclerosed carotid artery. The experiences of the pathologist are that lesions of the visual pathway are caused chiefly by arteriosclerotic softenings of varying size and only exceptionally by mechanical compression of a part of the pathway by an arteriosclerotic vessel. This experience applies especially to descending primary atrophy of the optic nerve. Arteriography (roentgen examination of the skull after the injection of colloidal thorium dioxide into one carotid artery) may give valuable information in cases of cerebral vascular disease, but is permissible only in carefully selected cases.

P. C. KRONFELD.

Ocular Muscles

FUNCTIONAL TREATMENT OF STRABISMUS WITH ELIMINATION OF AMBLYOPIA AND CORRECTION OF BINOCULAR VISION BY PRISMATIC SPECTACLES. H. E. ZIMMERMANN, Klin. Monatsbl. f. Augenh. **100:** 600 (April) 1938.

Binocular vision was considerably disturbed in 148 of 150 patients with strabismus, 122 of whom presented the unilateral convergent type,

20 the alternating convergent type and 8 the divergent type. Sixty-eight of the patients had disturbing amblyopia, which in 18 amounted to less than 1/60 of the normal vision. In most instances the angle of squint was greater in amblyopia of a high than in that of a low degree. Complete recovery from the amblyopia was obtained by occlusion of the better eye in 46 of 50 amblyopic patients with central fixation and in 16 of 18 patients with excentric fixation. Recurrences could be prevented, and depth vision was regained in a few cases by correction with prisms or liberal decentration of the glasses or by surgical procedure. Of 27 patients who had neither depth vision nor fusion, 10 obtained completely successful results, while 15 others obtained only fusion.

K. L. STOLL.

Operations

POSTERIOR SCLEROTOMY AS A FORM OF TREATMENT IN SUBCHOROIDAL EXPULSIVE HEMORRHAGE. D. VAIL, Am. J. Ophth. **21**: 256 (March) 1938.

Vail reports 2 cases of subchoroidal expulsive hemorrhage in which posterior sclerotomy of the temporal side of the globe gave successful results.

W. S. REESE.

AN EFFECTIVE OPERATION FOR ENTROPION IN TRACHOMA. F. HARBERT, Am. J. Ophth. **21**: 268 (March) 1938.

Harbert performed partial tarsectomy in 30 cases of trachoma, with good cosmetic and functional results. The operation is based on the idea of transplanting the margin of the lid to a higher level. About three fourths of the tarsus is removed, a strip being left below to preserve the margin of the lid. The conjunctiva, which is dissected from the tarsus, is used to cover the defect resulting from the removal of the tarsus.

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

THE INTRANASAL APPROACH FOR REMOVAL OF CERTAIN ORBITAL TUMORS. E. STIEREN, Pennsylvania M. J. **41**: 892 (July) 1938.

Stieren reports a case of osteoma of the orbit in a woman aged 48. There was exophthalmos on the left side, with homonymous diplopia in all cardinal directions. Movements of the globe appeared full. Neither the vision nor the fundus picture is recorded. Roentgenograms revealed a bony tumor arising from the nasal wall of the orbit and projecting forward.

Schlesinger and McKee removed the growth by performing a submucous resection; the ethmoid bones and middle turbinate were removed, exposing the orbital plate. This was broken through, and the tumor was crushed, as it was too large to be removed through the opening. Histologically, the base was composed of cancellous bone and the remainder of compact bone. The nasal route is suggested for the removal

of cancellous bony tumors of the orbit which arise in the ethmoid sinus and are accompanied by disease of the sphenoethmoid region, allowing correction of both conditions with one operation.

W. ZENTMAYER.

Physiology

DETERMINATION OF THE PRESSURE IN THE INTRAOCULAR ARTERIES. H. SERR, Arch. f. Ophth. **138:** 893 (July) 1938.

The development of dynamometry and the technic of, and the errors involved in, this method are again presented from the point of view of the Seidel school. Dynamometry is, in principle, not suitable for the determination of the pressure in the intraocular arteries because the process profoundly alters the pressures which are to be measured. The application of pressure from the outside raises the intraocular tension whereby the blood flow in all vessels in which the pressure is lower than the raised intraocular pressure is slowed down or even brought to a standstill, until through the vis a tergo from the arterial side the pressure in the vessels rises to the level of the raised intraocular pressure and circulation continues at a new level. This interference with venous and capillary circulation during application of the dynamometer results in incorrectly high readings for the arterial pressure. This error due to damming of the circulation (*Stauungsfehler*) is greater if the test is done slowly than if it is done quickly. By doing it quickly, Bailliart obtained for the intraocular arteries pressure readings which agreed closely with the readings which Seidel obtained for the anterior ciliary arteries with his "unobjectionable" method of applying a transparent pressure chamber to their epibulbar branches. Since the pressure in these anterior ciliary arteries must be in the neighborhood of that prevailing in the intraocular arteries, results obtained by dynamometry can be correct only if they are in agreement with Seidel's results for the anterior ciliary arteries (from 30 to 40 mm. diastolic; from 60 to 70 mm. systolic). Higher readings in the absence of cardiovascular hypertensive disease and theories based on such high readings (Sobański, from 48 to 56 mm. diastolic; from 80 to 90 mm. systolic) are of doubtful value.

P. C. KRONFELD.

Retina and Optic Nerve

MELANOSIS RETINAE. G. CIOTOLA, Ann. di ottal. e clin. ocul. **66:** 543 (July) 1938.

Forty cases of melanosis retinae are recorded in the literature. Two typical cases are reported. One was that of a girl of 15 years and the other that of a woman of 26. Both patients had normal vision and normal visual fields. The only ocular findings were the peculiar areas of grouped pigment deposits in the deeper layers of the retina. The retinal vessels and the retina around and between the spots were normal. In the first case both eyes were affected; in the second case, only the right eye. In nearly all of the reported cases the condition has been stationary with no effect on vision. The only report of sections is that of Parsons. who found an accumulation of cells in the pigment epithelium in the

affected areas. The author reports on sections of an eye removed for melanosarcoma. The fundus had not been observed during life, but aside from the tumor, a marked thickening of the pigment epithelium in certain peripheral portions of the retina was present, which he believes may represent the same condition. He points out that the condition is in the retina itself and is to be differentiated from melanosis of the choroid, although in the sectioned eye this condition was also present.

Drawings of the fundi and sections accompany the article.

S. R. GIFFORD.

CENTRAL DETACHMENT OF THE RETINA OBSERVED FOR A LONG PERIOD: REPORT OF CASES. S. PREVEC, Klin. Monatsbl. f. Augenh. **100:** 222 (Feb.) 1938.

Central detachment of the retina occurs more rarely than the peripheral type. Observation of cases of this type of detachment reveals distinct differences from other types. Prevec was able to study in 4 patients the development of a central detachment, beginning with cystoid foci in the macula followed by the formation of a central hole and progression to complete detachment. Two of the patients furnished especially good opportunities for extended observation by their refusal to submit to an operation. The smallness of holes in the macula explains the confusion of these holes with macular cysts. The 4 patients, aged 68, 63, 72 and 77, respectively, presented senile degenerative changes in their eyes, which were hyperopic. Extensive detachment rarely follows the formation of a hole in the macula, because the retina is thickened in the area of the macula and because the absence of traction of the vitreous will not lead to protrusion of the edges of the hole. Prevec observed in 1 of his patients that the development of new cysts around an originally circular hole in the macula produced final closure. The vitreous in these cases need not be pathologically deteriorated, hence detachment of the vitreous was noted in 1 case only. Dehiscence of the retina occurs along blood vessels and nerve fibers; their absence in the macular region tends to prevent the spread of a detachment. The effect of the jerking motion of the subretinal fluid is limited in the macular area. Prevec expresses the opinion that detachment of the macular region may be treated conservatively in many instances.

K. L. STOLL.

RENAL FUNCTION IN DIABETIC RETINITIS. S. HANUM and K. B. MORTENSEN, Acta ophth. **16:** 396, 1938.

In order to obtain an expression of the functional efficiency of the kidneys of patients with diabetic retinitis, the authors made determinations of the creatinine, urea and uric acid clearance for a series of 20 such persons. (Clearance of a substance means the number of cubic centimeters of blood which contains the quantity of the substance that is excreted through the kidneys in one minute.)

The results obtained showed no reduction in the clearance of these substances, and no retention of urea was found except in 1 patient who also had nephrosclerosis.

O. P. PERKINS.

Uvea

LATE RESULTS OF OPERATION IN CASES OF IRIDOCYCLITIS. A. MIKLÓS, Arch. f. Ophth. **139:** 403 (Sept.) 1938.

This report is based on material from Professor Kreiker's clinic in Debrecen, Hungary. Only data on patients who were followed for years after operation have been included. In cases of diffuse tuberculous iridocyclitis iridectomy not only does not stop the progression of the disease but may even influence its course unfavorably. In such cases iridectomy should be performed only if the presence of secondary glaucoma forces one to operate. In cases of chronic recurrent iridocyclitis of rheumatic origin the situation is different. Iridectomy may cause a definite improvement in the course of the disease. Puncture of the anterior chamber seems to be more effective in the acute and subacute forms of iridocyclitis than in the chronic forms. When an operation is to be performed for uveitic (complicated) cataract, the intracapsular procedure is preferable to the extracapsular method.

P. C. KRONFELD.

Therapeutics

GENERAL CONSIDERATIONS IN THE RADIATION TREATMENT OF SKIN CANCER IN THE REGION OF THE EYE. W. L. WATSON and W. WUESTER, Am. J. Ophth. **21:** 261 (March) 1938.

Watson and Wuester discuss the treatment of cancer of the skin in the region of the eye with roentgen rays and gold radon seed. They give the following summary:

"1. Various considerations in regard to the treatment of skin cancer in the region of the eye are discussed. Low voltage x-ray is the means of choice, advocated for its efficiency and for the safety factors and economic considerations offered.

"2. No dosage factors are given, for each case presents its own specific problem of management."

W. S. REESE.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W. WHITE, M.D., *Chairman*

Jan. 16, 1939

RUDOLF AEBLI, M.D., *Secretary*

MENINGIOMA PRODUCING UNILATERAL EXOPHTHALMOS. DR. JAMES W. SMITH.

This article will be published in full in a later issue of the ARCHIVES.

A CALIBRATED NIPPLE FOR CONTACT LENSES. DR. JOSEPH I. PASCAL.

This article will be published in full in a later issue of the ARCHIVES.

KERATITIS IN HURLER'S DISEASE (GARGOYLISM). DR. MILTON L. BERLINER.

This article will be published in full in a later issue of the ARCHIVES.

A RARE TUMOR OF THE ORBIT. DR. WILLIAM MCLEAN.

A girl, aged 15 years, was hospitalized because of a diseased area of the spine associated with fracture. She had also an atypical tumor-like growth in the left retrobulbar space, with proptosis. The coexistence of an adenopathy of the left cervical glands of the same histological nature, with the blood triad of progressive anemia, persistent leukocytosis, associated with myelocytes and myeloblasts and accompanied by a myeloblastic hyperplasia, and thrombocytopenia, would appear to indicate an acute myeloblastic leukemia with characteristic infiltrations of the spine, orbit and cervical nodes and possibly of other tissues.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Chairman*

Feb. 16, 1939

WARREN S. REESE, M.D., *Clerk*

ADDITIONAL RESEARCH ON VERNAL CONJUNCTIVITIS. DR. LOUIS LEHRFELD and DR. JEROME MILLER.

This article appeared in full in the April issue of the ARCHIVES, page 639.

DISCUSSION

DR. LUIS TUFT: The authors are to be commended for continuing their studies and furnishing additional evidence of the allergic character

of vernal conjunctivitis. The seasonal incidence, the presence of a mucoid discharge containing many eosinophils and the occurrence of a positive cutaneous reaction in a high proportion of cases were described in a previous study. And now the finding of a positive family history or a personal history of allergy in a fairly high percentage of cases and the occurrence of a positive ocular reaction to dry pollen furnish additional proof of the allergic character of this condition.

Vernal conjunctivitis cannot be accepted as truly atopic, however, at least not in the same sense as is true of hay fever or asthma. In the latter disease the hereditary factor is more definite and is found in a higher percentage of cases. Cutaneous reactions are more specific and correspond more definitely to the patient's clinical condition, and specific allergic antibodies or reagins can be found in the blood of the patient. This is not true as yet of vernal conjunctivitis.

The only difficulty in accepting vernal conjunctivitis as truly allergic is in the determination of a specific etiologic agent. It is true that positive reactions to pollens or other inhalants are obtained with cutaneous and ocular tests, but this is not enough to account for the symptoms which, extending as they do from spring throughout summer, do not seem to be due to any one pollen, as in hay fever, or even to several pollens. Most patients with hay fever are sensitive to one group of pollens. The symptoms are limited to one season and do not extend through several seasons. Even patients who are sensitive to such inhalants as dust, orris and tobacco and who have conjunctivitis do not have the vernal type of conjunctivitis, and yet they give positive reactions to cutaneous tests.

Because of the uncertainty as to the cause of vernal conjunctivitis, it occurred to me that possibly sensitivity existed not to the aqueous fraction of a pollen (the fraction responsible for hay fever) but to an oily fraction (the fraction responsible for pollen dermatitis) and that vernal conjunctivitis resulted from contact of sensitive persons with this fraction. This might account for prolonged action over a long period. I suggested that tests with the oily fraction both by the patch method and in the eye be included in the study. Negative reactions to patch tests were obtained, but the pollen oil proved too irritating in the eye for satisfactory study, so that no evidence of sensitivity to pollen oil was obtained in the present investigation.

Other suggestions as to the cause of vernal conjunctivitis include possibility of sensitivity to molds and also possibility of bacterial sensitivity, the latter being suggested by Dr. Cooke. These are theoretic and still await experimental confirmation. It is apparent, therefore, that although evidence has been presented to warrant the conclusion that vernal conjunctivitis is allergic, its causation is undetermined. The same difficulty exists, possibly, with vernal conjunctivitis as with certain cases of allergic rhinitis. Many of the patients with allergic rhinitis have seasonal symptoms and are recognized as having hay fever the cause of which is determinable; others have perennial symptoms, and here again the cause is either sensitivity to inhalants or to foods; finally, there is a small but definite group of persons who have symptoms of allergic rhinitis which begin in the fall and last throughout the winter and spring and clear up in warm weather. These patients also give positive cutaneous reactions to various allergens, but the reactions are

not specific and treatment for specific allergy is not followed by improvement. It is possible that these persons have what Duke has called physical allergy, or sensitivity to physical agents. Possibly vernal conjunctivitis, too, represents a similar condition, a type of conjunctivitis in between that of hay fever on the one hand and perennial allergic conjunctivitis due to dust or other inhalants on the other. It might be well to insist that patients with vernal conjunctivitis be treated by allergic methods in order to determine whether they respond to such treatment in the same way as other allergic patients.

DR. JEROME MILLER: We have presented presumptive evidence which indicates that vernal conjunctivitis resembles in many ways other allergic diseases. This was manifested by the seasonal incidence, the eosinophilia in the blood and ocular discharge, the positive intradermal and ophthalmic reactions to the various allergens and pollens and the symptomatic relief afforded patients by the use of a solution of hydrochloride epinephrine.

While vernal conjunctivitis is not an atopic disease in the sense that hay fever and asthma are, it is subject to the influence of heredity. We have shown that heredity plays an important part in this disease and that the factor of age corresponds to the age incidence of other atopic states. Vernal conjunctivitis in our series of patients was shown to exist in the same family and also in members of families in which other atopic diseases were present.

Having definitely established the fact that vernal conjunctivitis is an allergic disease, our future work will be directed toward determining the specific allergen or allergens in question and any other factor, whether specific or secondary, which may cause a prolongation of symptoms.

MANAGEMENT OF HYPERMETROPIA. DR. ALFRED COWAN.

In the management of hypermetropia careful examination under cycloplegia as well as after cycloplegia is important. The findings with the postcycloplegic test are particularly important as factors bearing on the final prescription for glasses.

EXTENSIVE ULCERATION OF THE CORNEA CLEARED BY DESICCATION PERITOMY: REPORT OF A CASE. DR. VAN M. ELLIS.

A woman, aged 66, was first seen by me in April 1938, complaining of lacrimation, photophobia, pain and ability to see only large objects with the right eye.

Seven years previously she had suffered from occasional pain in the right eye, sensitivity to light and blurred vision at a distance. Tearing and cloudy vision became progressively worse. For the past four or five years she had been unable to see anything distinctly with the right eye. The past history was otherwise essentially unimportant.

On examination the right eyelids were red and swollen and the eye closed. The palpebral conjunctiva was much inflamed. Numerous vessels from the conjunctiva crossed the limbus onto the cornea in its entire circumference. The cornea was bedewed and hazy throughout. There was superficial staining. Slit lamp examination showed that the

cornea was thickened, and there were many deep folds in Descemet's membrane. The anterior chamber appeared to be of normal depth. Pupillary reactions were normal. Details of the iris could not be clearly seen. Vision was reduced to perception of hand movements. Examination of the left eye, externally and ophthalmoscopically, gave negative results. Vision was 6/6.

A general clinical examination, including Mantoux and Wassermann tests, blood sugar tests, examination of the teeth and nose and throat, gave negative results. Treatment consisted of a diet high in vitamin content, the administration of salicylates and ethylmorphine hydrochloride, the use of hot compresses and the application of a 1 per cent solution of silver nitrate to the lids. An eye patch was applied. This treatment was continued without any apparent improvement.

On September 9 a desiccation peritomy was done, after which the vision began slowly to improve. In one month vision improved to 6/15, and bedewing and general appearance of the cornea had changed considerably. On October 17 many of the vessels that had been missed at the first desiccation were touched individually. During the following three months the cornea continued to clear, and the vision gradually improved.

On Jan. 31, 1939, several small fine remaining vessels were desiccated. Vision on February 15 in the right eye with a + 0.25 sph. ⌒ — 0.75 cyl., axis 170 was 6/9; with the addition of a + 2.50 sph. for near vision, the patient was able to read 0.50 meter test type.

The desiccation method of interrupting the blood vessels at the limbus has been used in this case of ulcerative keratitis with extreme vascularization of the cornea. Considering the chronic nature of the condition, the result speaks for itself, leaving no doubt that desiccating peritomy is of great value in many corneal conditions in which there is vascularization.

DISCUSSION

DR. H. MAXWELL LANGDON: In earlier days when there was much immigration and patients with trachoma eluded medical inspection, peritomy for the vascularity of the cornea was a frequent proceeding. Trachoma is now of infrequent occurrence, and peritomy has fallen into disuse; however, it is most helpful for a vascular corneal ulcer.

ARGYLL ROBERTSON PUPIL, APPARENTLY NONSYPHILITIC: REPORT OF THREE CASES. DR. H. MAXWELL LANGDON.

In the *Edinburgh Medical Journal* for February 1869 there appeared an article by Argyll Robertson on "An Interesting Series of Eye Symptoms in a Case of Spinal Disease."

The case was that of a man 59 years old who had small unequal pupils which contracted in accommodation but not to light stimulation and which would not dilate well with atropine. The optic nerves were atrophic. The miosis was of chief interest to Argyll Robertson in this case, and he attributed it to weakness of the sympathetic dilating fibers from the spinal cord.

In a second article in the December 1869 issue of the same journal he reported 4 more cases and gave much more consideration to the subject of the response of the iris to light and in accommodation.

In 3 of these cases there was atrophy of the optic nerve, and in all of them the pupils were inactive to light but responded in accommodation. He directed special attention to the phenomenon but had no satisfactory explanation for it. This pupillary condition, which has become known as the Argyll Robertson pupil, was finally found to be present most frequently in diseases of the spinal cord and, with the absence of knee jerks and a positive Romberg sign, has become diagnostic of locomotor ataxia. As this syndrome is now known to be due to syphilis, it has become a sign of neurosyphilis, although it may also be present in epidemic encephalitis and in posterolateral sclerosis.

In the *Transactions of the Ophthalmological Society of the United Kingdom* for 1924 R. Foster Moore reported 7 cases of "nonsyphilitic Argyll Robertson pupils," and in the same publication in 1931 he added reports of 8 more cases, making 15 in all.

The characteristic change in the pupils in this series was a complete loss of contraction to light but a response to accommodation-convergence stimulus, which was very slow both in contraction and in dilatation. Accommodation was not affected. Full dilatation was accomplished with atropine, and contraction followed instillation of physostigmine salicylate. The condition was unilateral in 13 of 15 cases reported, and the affected pupil was usually larger than the normal one. Twelve of the 15 patients were females, and the age of recognized onset was between birth and 42 years.

Changes were not found in any other ocular structure or in the central nervous system, and in no case could syphilis, either acquired or congenital be demonstrated.

In the *British Medical Journal* for May 1931 Adie reported 6 cases of pseudo Argyll Robertson pupils of the type which Foster Moore had already reported. Two of the patients had been referred to him by Moore, and the reports of their cases have apparently been included in Moore's series.

The following 3 cases seem to me to be of the type described by Moore and Adie but to be more representative of the ordinary manifestations of pupillary changes in disease of the posterior part of the spinal cord, and yet 1 patient has been under observation for thirteen years and another for fourteen years without showing any of the general manifestations. The third patient, who has been only recently observed, shows no general symptoms or serologic changes to justify a diagnosis of neurosyphilis.

Mr. M. P. B., aged 52, was seen by me on June 17, 1926, through the courtesy of Dr. Malcolm Councill. It had been noticed about two weeks before that the pupils were unequal and did not respond to light. The patient was in good health in every way, and Dr. Councill could find no neurologic symptoms. Examination showed that with his low hyperopia corrected his vision was 5/4 in each eye and his near point with a + 2 sphere was suitable for his age. The right pupil was 3.5 mm. in diameter and the left 5 mm.; there was no response to light and rather slight contraction in accommodation. The power of accommodation, however, was preserved. Ocular rotations were not impaired, and there was no diplopia. The media were clear, and the disks were normal in color. There was some broadening of the light streak of the retinal

arterioles but no other changes in the fundi. The patient refused to have any serologic tests made. At the time of this report he is still in perfect health, but the pupils are unchanged. No attempt was made to dilate the pupils with mydriatics.

On May 13, 1925, Mr. W. W. L., aged 35, was seen, complaining of some dimness of vision for close work. His vision for distance was clear. He had no other ocular symptoms and no headache. The vision of the right eye was 5/6 +, and the near point was 21 cm.; the vision of the left eye was 5/5 +, and the near point was 19 cm. The right pupil was 4 mm. in diameter, was irregular in outline and did not respond even to concentrated light. The left pupil was 3 mm. in diameter, was irregular in outline and responded slightly and slowly to diffused light. The pupils responded well to accommodation-convergence stimulation. Ocular rotations were normal, and there was no diplopia. The visual fields were normal for form and color. The media were clear, and there were no changes in the fundi. The Wassermann reaction of the blood was negative. The patient refused an examination of the spinal fluid. He has been seen within six months and seems to be in perfectly good health. So far as he knows, there is nothing the matter with him in any way. The pupils are still unchanged. No attempt was made to dilate the pupils with mydriatics.

On Nov. 5, 1938, Miss E. A. M. was seen through the courtesy of Dr. W. E. Robertson. She gave a history of trouble in using her eyes, and Dr. Robertson had noticed that the pupils did not react well to light. Her eyes felt strained for near work, and she had occasional headaches. Vision was 5/4 both with and without glasses. Her near point was 18 cm. The right pupil was 3.5 mm. in diameter and the left 3 mm. The pupils were irregular in outline and did not react even to concentrated light, but they responded moderately well to accommodation-convergence stimulation. The pupils dilated poorly with homatropine hydrobromide. The media were clear; the disks were normal in color and outline, and there were no changes in the fundi. The visual fields were normal for form and color. There was no diplopia. Dr. Robertson was unable to find any clinical signs which would justify a diagnosis of disease of the central nervous system, except for slightly sluggish knee jerks. The Meinicke and Wassermann reactions of the blood were negative. The spinal fluid was perfectly clear. The Meinicke and Wassermann reactions of the spinal fluid were negative; the reaction to the colloidal gold test was 0000000000, and the reaction to the globulin test was negative.

DISCUSSION

DR. FRANCIS HEED ADLER: In my opinion the condition in these cases fits into Adie's syndrome rather than into the classification of true Argyll Robertson pupils. The pupils were not myotic, and at least 1 patient definitely showed tonic reaction.

DR. WALTER I. LILLIE: Was a mydriatic or a cycloplegic used to ascertain whether or not the pupils would dilate?

DR. H. MAXWELL LANGDON: The pupils of 1 patient did not dilate well with homatropine hydrobromide, and it was not used for the other 2 patients.

Book Reviews

Patologia e chimica della regione diencefalo-chiasmatico-ipofisaria, con particolare riguardo alla sintomatologia oculare. By Dr. A. Rubino. Price, 30 lire. Paper. Pp. 242, with appendix and bibliography. Padova, Cedam, casa edit. dott. Antonio Milani, 1937.

This monograph is thoroughly practical in its scope and accomplishes the author's purpose of uniting in one volume all conditions which are known to have their origin in the "diencephalic-chiasmic-hypophysial" region. To make the material more available, Rubino has adapted an almost outline form of writing.

While the title states that special reference is made to ocular symptoms, the value of the volume is increased by the fact that equal space is devoted to the role of special examinations in developing a more exact differential diagnosis of this highly complex region. Great emphasis is placed on the need for close cooperation between the ophthalmologist, the internist, the neurologist and the roentgenologist.

The first part of the monograph is composed of two chapters, the first of which is a review of the anatomy of the region. The second deals with the clinical signs, both general and ocular, and with the method of carrying out, and the value of, specialized technics. The remaining portion, of eight chapters, is given over to a systematic discussion of the diseases involving the chiasm and its surrounding structures. A short review of the literature is given for each disease and is followed by a description of the general and ocular signs and symptoms. The findings obtained by special technics, such as roentgenography, ventriculography, encephalography, cerebral angiography and lumbar puncture, are presented when they are significant. Finally, the differential diagnosis is reviewed in each case.

A short appendix containing criteria for therapy is added. Inflammatory lesions should be treated medically. All tumors of the hypophysis and the surrounding structures should be treated surgically at the earliest time after a definite diagnosis. The only exception is the chromophilic tumor of the hypophysis, which may be treated with roentgen rays. In chiasmic arachnoiditis, as soon as the visual acuity drops rapidly the visual field shows a continuous constriction, and when signs of atrophy of the optic nerve are noted surgical intervention is indicated immediately.

A valuable extensive bibliography is given. There is little doubt that this volume would be a worth while addition to the library of every ophthalmologist, neurologist, internist and roentgenologist.

F. P. Guida.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.
Place: Aberdeen, Scotland. Time: July 27-28, 1939.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: Sept. 4-6, 1939.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 81 Edmund St., Birmingham, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Time: July 6-8, 1939.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome, Italy.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome, Italy.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: New York. Time: June 10-14, 1940.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: Dr. Edward Jackson, 1008-A Republic Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 6:30 p. m., third Saturday of each month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. C. Smith, 106 W. 7th St., Charlotte.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Place: Statesville. Time: Sept. 21. 1939.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.
Place: Fargo. Time: May 1939.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Don Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St., N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 58 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW HAVEN OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Pl., Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Ernest Sheppard, 927 Farragut Sq., N. W., Washington, D. C.
Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St., N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

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